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ANALYSIS OF OPERATIVE RESULTS IN CONCOMITANT CONVERGENT STRABISMUS

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There are two particularly important decisions which have to be made in regard to operations for concomitant convergent strabismus. The first and most important of these is the selection of the proper muscles for the operation, and the second is the determination of the amount of operative correction which should be done. In an attempt to improve the results of such operations two series of patients were operated on by members of the ophthalmic staff. The patients comprising the first series were subjected to bilateral recession of the internal rectus muscles. For those patients in the second series unilateral recession of the internal rectus muscle was combined with resection of the external rectus muscle of the same eye. On completion of these series of operations, analysis of the preoperative, operative and postoperative data was undertaken. From this material some general principles have been derived which may be of assistance in the solution of future problems. When operative measures are indicated, it is necessary to select the proper type of procedure to meet the demands of the muscular abnormalities in the particular case. This selection implies the proper use of those surgical procedures which diminish and those which increase muscular action. It is also necessary to apply these two types of procedures to the four main horizontally acting muscles. It follows that the correct application of the two main types of surgical procedures to the four main horizontally active muscles requires a knowledge of the functional status of the muscles in the particular case. This is necessary so that muscles which are functioning excessively may be subjected to a procedure that will diminish their action and so that those muscles the action of which is insufficient may be subjected to the procedures that will improve their action. Furthermore, it is necessary that these various procedures be properly graduated in amount in each individual case.

From the Department of Ophthalmology, Temple University School of Medicine.

Read before the College of Physicians of Philadelphia, April 20, 1939.

A discussion of this article appeared in the transactions of the society, in the September issue of the ARCHIVES (22:511, 1939).

The material for this study was composed of 66 patients, of whom 44 were operated on by the bilateral recession technic and 22 were operated on by the recession-resection procedure. The youngest patients (4) in this group were 4 years of age, and one fourth of the entire group were under 8 years of age. The rest of the patients were between 8 and 25 years of age. Since the deviation was present for two years or more in all these cases at the time of the first examination, the various sequelae of strabismus were well established. In view of this, a complete cure of the sequelae was neither attempted nor achieved. For these reasons the main surgical objective was a cosmetic improvement rather than the more ideal establishment of binocular single vision. The operative procedures in each series were done on consecutive patients, and selection of the procedure was not done as a rule. This plan of procedure was carried out for comparative purposes.

The purpose of this report is to give the results of the analysis of this group of operations. The operative treatment for early strabismus is not included in this report. No attempt is made to report new or modified procedures or to discuss the relative merits of various types of operations on the muscles. The literature is so extensive on this subject that it has not been included, but it is well discussed in such recent publications as those of Wheeler,[1] Spaeth,[2] Jameson[3] and Prangen.[4]

The amount of strabismus was determined by the Hirschberg method, in which the position of the corneal light reflex is utilized. Figure 1 is a controlled photographic representation of the position of the corneal reflex as it occurs in the various degrees of deviation. This method was selected because of the simplicity of the determination, and the accuracy of this test correlates the accuracy of the surgical procedures. The factors which introduce variation in this test are omitted from this article. Symmetrically placed corneal reflexes were the criteria used to determine that the eyes were straight. Some of the eyes which were recorded to be straight by this test were slightly eccentrically placed when measured by a telescopic stereoscope. The postoperative results which are presented are not the immediate results but represent the latest records obtainable.

The patients were divided into those with minimum, moderate, marked and maximum grades of strabismus (grades 1, 2, 3 and 4),

1. Wheeler, M. C.: Surgical Treatment of Strabismus: Review of Recent Literature, Arch. Ophth. **18**:1000-1010 (Dec.) 1937.

2. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, Philadelphia, Lea & Febiger, 1939, chap. 6.

3. Jameson, P. C.: Entity of Muscle Recession: A Short Résumé of Its Technic and Principles with New Supplementary Notes and Illustrations, Arch. Ophth. **21**:362-370 (Feb.) 1939.

4. Prangen, A. de H.: Surgery of the Rectus Muscles of the Eye: Selection of Operative Procedures by Differential Diagnosis, Arch. Ophth. **18**:151-156 (Feb.) 1935.

according to the amount of the deviation. This classification is helpful both clinically and analytically and is as follows:

Grade 1.....10 to 20 degrees
Grade 2.....21 to 35 degrees
Grade 3.....36 to 45 degrees
Grade 4..... over 45 degrees

The determination of the amount of excessive or diminished excursions of the eyeball in the extremes of the horizontal plane was used to evaluate the functional condition of the four horizontal rectus muscles. The various amounts of excessive and diminished rotation are represented diagrammatically in figure 2. When the eyeball moved nasally farther than normal, the internal rectus muscle was considered to be

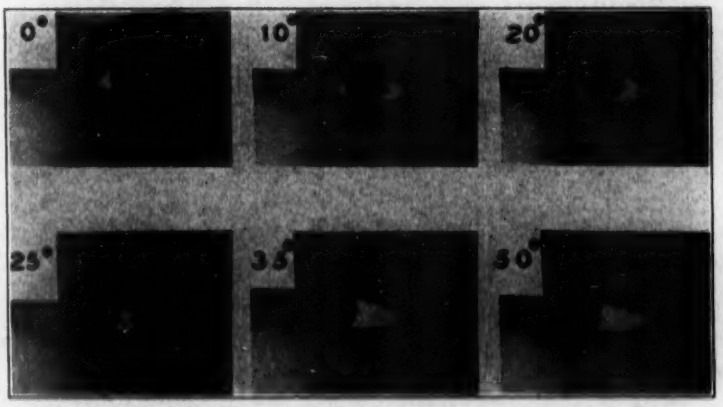

Fig. 1.—The position of the corneal reflex on the deviating eye in the various degrees of strabismus as determined by the Hirschberg method.

functioning excessively. This excessive rotation was classified as +1, +2, +3 or +4, depending on the amount and the speed of the excessive movement of the eye. When the temporal rotation of the eyeball was less than normal, the external rectus muscle was considered to be functioning insufficiently. This insufficiency of rotation was classified as —1, —2, —3 or —4, depending on the degree and the hesitancy of the insufficient rotation. When the motility of the four rectus muscles is properly classified, a comprehensive conception of the abnormal rotations of each eye is obtained. The classification of muscular action is of assistance in deciding, first, which muscles should be selected for operation and, secondly, the distribution of the operative correction which is required. This information makes it possible to operate in accordance with the fundamental principle of determining the amount of correction and the type of procedure by the underlying pathologic muscular condition present in the individual case.

The preoperative and the postoperative distribution of the number of cases of strabismus and of the amount of deviation present in the group in which bilateral recession was done is graphically represented in figure 3. From this graph it is apparent that the majority of patients obtained satisfactory results. It further shows that certain patients were materially improved but did not obtain complete correction and that a smaller group obtained overcorrection. A similar distribution is shown in figure 4, which represents the results of the recession-resection technic.

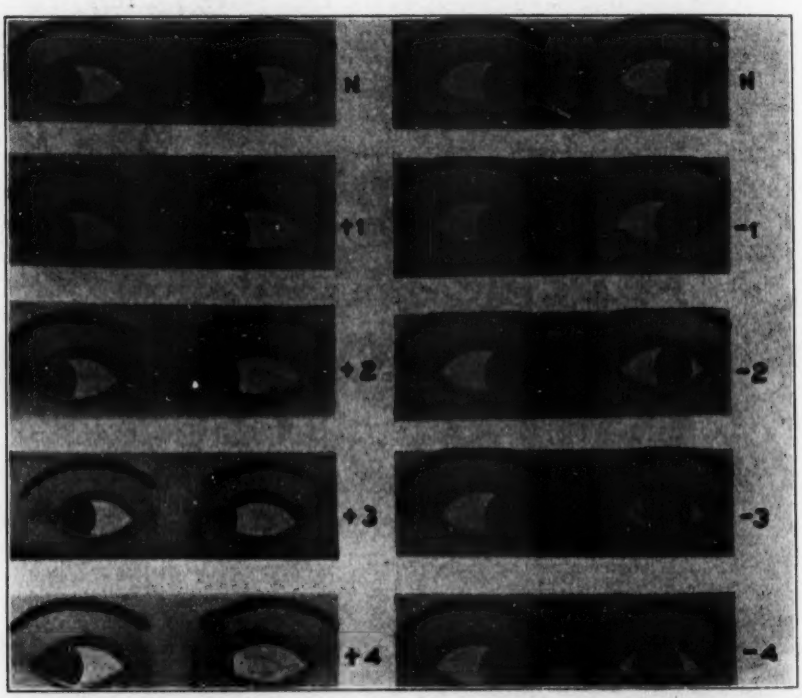

Fig. 2.—The first column shows the normal position of the right eye in eyes right. The left eye is shown in the four grades of hyperaction of the left internal rectus muscle. The second column shows the normal position of the right eye in eyes left. The left eye is shown in the four grades of insufficiency of the left external rectus muscle.

The distribution of the results according to the grade of the strabismus (table 1) shows that patients with strabismus of grade 1 responded better when operated on by the bilateral recession technic than by the recession-resection procedure. Those with strabismus of grade 4 responded more satisfactorily to recession and resection than to bilateral recession. Overcorrection occurred only in cases of strabismus of grades 1 and 2.

The distribution of the results according to the visual acuity (table 2) reveals that the patients with amblyopia ex anopsia responded equally well when operated on by either procedure. The amblyopic patients

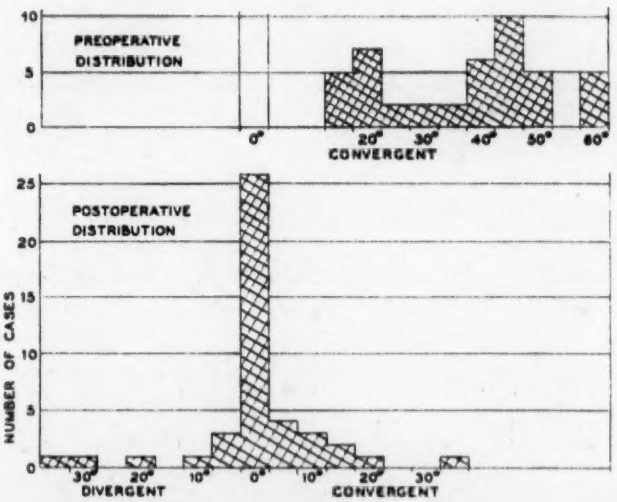

Fig. 3.—Graph showing the distribution of the number of cases of strabismus and the amount of deviation before and after bilateral recession in a group of 44 cases.

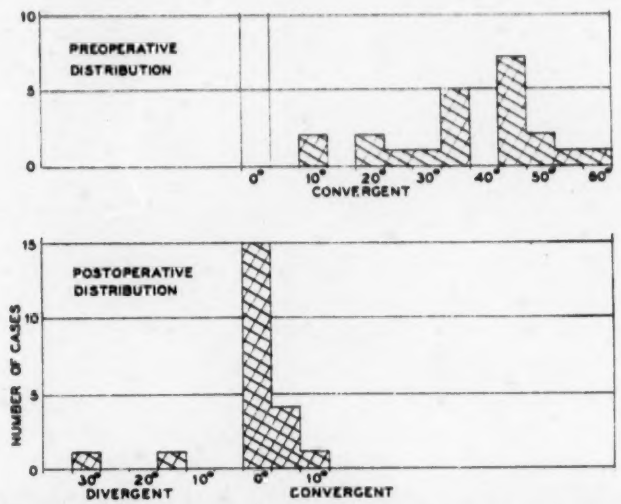

Fig. 4.—Graph showing the distribution of the number of cases of strabismus and the amount of deviation before and after recession combined with resection in a group of 22 cases.

attained a higher percentage of correction than was attained by persons with good vision in each eye.

The amount of operative correction which produced ocular parallelism most frequently in the various degrees of strabismus is given in figure 5. This graph was derived by determining the amount of total operative correction which most frequently resulted in correction for each of the various degrees of strabismus. The graph illustrates four main grades of operative procedures which are applicable to the four corresponding grades of strabismus. Patients with strabismus of 10 to 20 degrees did best when subjected to from 4 to 6 mm. of operative correction, divided between the two muscles. Patients with strabismus of 25 to 35 degrees responded more satisfactorily when they received 6 to 8 mm. of total operative correction, while those with 40 to 50 degrees of

TABLE 1.—*Distribution of the Results According to the Grade of Strabismus*

	Recession-Resection					Bilateral Recession				
Result	Grade 1	Grade 2	Grade 3	Grade 4	Total Cases	Grade 1	Grade 2	Grade 3	Grade 4	Total Cases
Straight	1	6	4	4	15	5	5	10	6	26
Undercorrection	1	1	3	0	5	1	2	4	4	11
Overcorrection	2	0	0	0	2	2	4	1	0	7
Total	4	7	7	4	22	8	11	15	10	44

TABLE 2.—*Distribution of the Results According to Visual Acuity*

	Recession-Resection		Bilateral Recession	
Result	6/10 + O. U.	Amblyopia	6/10 + O. U.	Amblyopia
Straight	2	11	12	10
Undercorrection	1	4	6	2
Overcorrection	0	2	1	1
Total	3	17	19	13

convergent strabismus attained correction more frequently following 9 to 10 mm. of total operative correction. Twelve millimeters of total correction, divided between the two muscles was necessary to overcome a convergent strabismus of more than 50 degrees. Under identical conditions more correction is usually attained by recession-resection operations than by bilateral recession. Table 2 represents the average amount of correction which will be required in the usual case. This amount of correction will not always produce parallelism, as there are exceptions to these general principles. The reason for the variability in the results is that the effect of the procedure is determined not only by the amount done and by the condition of the muscle on which operation is performed but by the functional condition of the other three horizontal rectus muscles (and occasionally by the condition of the muscles which play a secondary part in the horizontal movement).

It is important to compare the relative rotational power of the muscles of the two eyes. The two main distributions of abnormal rotation encountered were (1) those in which there was an equal amount of rotational disturbance in each eye and (2) those in which there was a unilateral preponderance of abnormal rotation. If the abnormal rotation is equal in the two eyes, the problem is different than it is when there is unilateral preponderance of abnormal rotation. When the rotations are essentially normal in one eye and abnormal in the other, the surgical procedures should be confined to the muscles of the abnormally rotating eye. The importance of this principle is illustrated in table 3. From this table it is seen that all 14 patients whose eyes were straightened by bilateral recession had equal excessive action of the internal rectus muscles. This demonstrates the importance of selecting a pro-

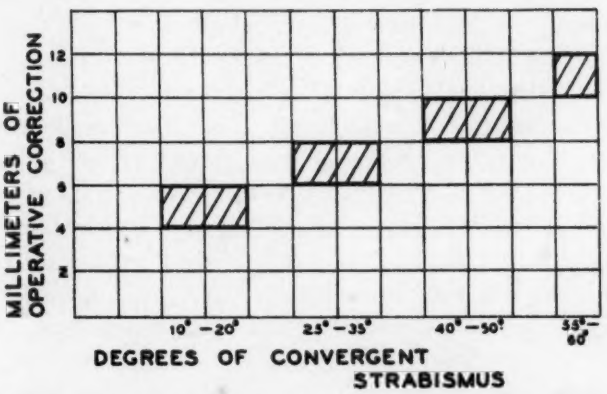

Fig. 5.—Graph showing the amount of total operative correction which succeeded most frequently in the cases of the various degrees of strabismus.

TABLE 3.—*Distribution of the Results According to the Type of Abnormal Muscular Action*

Muscular Abnormality	Recession-Resection			Bilateral Recession		
	Straight	Under-correction	Over-correction	Straight	Under-correction	Over-correction
Unilateral preponderance of abnormal rotations	8	2	0	0	2	1
Equal excess of internal rectus muscles	3	1	0	14	4	2
Equal insufficiency of external rectus muscles	0	0	0	0	2	2

cedure for patients with equally excessive internal rectus muscles which equally diminishes the excessive action of the hyperactive muscle. Those patients who had bilateral excessive action of the internal rectus muscles but whose eyes were not straightened by bilateral recession did not have

the amount of correction shown in figure 5. This demonstrates that not only is it desirable to use the proper procedure but it is also important to graduate the amount of the correction in proportion to the amount of strabismus and the amount of the muscular abnormality.

Likewise, 8 of the 11 patients whose strabismus was corrected by the recession-resection technic had unilateral preponderance of abnormal rotation. This indicates the importance of using a unilateral procedure in cases in which the abnormal rotation is mainly unilateral. Furthermore, patients with bilateral insufficiency of the external rectus muscles and normal rotation of the internal rectus muscles did not respond satisfactorily when subjected to bilateral recession. The rotational abnormalities are mainly due to abnormal innervation of the muscles or to muscular changes, such as hypertrophy, atrophy or contractures or a combination of these, and these factors must be taken into consideration whenever possible. The various causes of these abnormalities are beyond the scope of this analysis.

After the studies are completed, the plans for the operative procedures should be made in the examining room. Occasionally it is necessary to modify these plans on the operating table. The number of millimeters of total operative correction is determined by the amount of the strabismus. If grade 1 strabismus is present, usually a grade 1 operative correction is indicated, and so on for the other grades of strabismus. Exceptions to this rule are those instances in which unusually excessive muscular changes are encountered in the lesser degree of strabismus and in cases of strabismus grade 4 in which there is only slight abnormality of rotation. Smaller amounts of correction are indicated for the same amount of strabismus in children than is necessary for adults. After the number of millimeters of operative correction that should be used is determined, the next decision to be made is the distribution of the correction among the four horizontal rectus muscles. It is seldom desirable to attempt to obtain correction by limiting the operative measures in well established cases to one muscle, and it is rarely necessary to operate on more than two muscles at the time of the first operation. The total operative correction may be divided equally or unequally between the two muscles selected for operation. Since hyperactivity of the internal rectus muscles is usually demonstrable in concomitant convergent strabismus, it is usually necessary to diminish this hyperactivity by recession of one internal rectus muscle or of both of them. If this excessive action of the internal rectus muscles is equal and bilateral, an equal bilateral recession is the procedure of choice. If hyperactivity is unequally present and is not associated with insufficiency of the external rectus muscles, the total amount of recession should be unequally distributed in such a way as to apply most of the correction to the most hyperactive internal rectus muscle. This does not obtain in

cases in which there is over 50 degrees of strabismus, as it is usually necessary to depend on resection combined with recession in cases of grade 4 strabismus. If normal rotation of the internal rectus muscles is present, little if any recession is indicated. In those rather unusual cases in which the main rotational abnormality is an insufficiency of the external rectus muscles associated with fairly normal internal rectus muscles, the main reliance should be placed on resection of the former. The most common type of rotational disturbance encountered was a combination of a hyperactive internal rectus muscle with an insufficient external rectus muscle, and usually this abnormality is much more marked in the muscles of one eye. This is usually the amblyopic eye. The procedure of choice in this type of case is recession of the internal rectus muscle combined with resection of the external rectus muscle of the same eye. If the internal and external rectus muscles are equally abnormal in action, for example a + 2 internal rectus muscle combined with a — 2 external rectus muscle, the total operative correction should be equally divided between the two muscles. If, however, there is a preponderance of hyperaction of the internal rectus muscle, for example a + 3 internal rectus muscle and a — 1 external rectus muscle, the main surgical emphasis should be placed on the hyperactive muscle.

These statements are presented as general principles, because hard and fast rules are not justifiable in such a complicated problem as strabismus and on the basis of such a limited group of cases. In those instances in which the results were unsatisfactory, possible explanations for the outcome were: (1) unusually excessive or diminished muscular action, (2) various congenital ocular defects, (3) previous operations for strabismus and (4) such secondary muscular conditions as hypertrophy, atrophy and contractures.

The final decisions in the individual case should be based on the amount of strabismus, the rotational status of the muscles and the exceptional factors which complicate certain cases. It is most probable that by classification proper evaluation of the functional condition of the muscles, the selection of the appropriate muscles and the graduation of the amount of operative correction that it will be possible to reduce the number of instances in which secondary operations may be required in this group of cases in which the sequelae of the deviation are so well established that all efforts must necessarily fall short of the ideal.

CONCLUSIONS

1. Bilateral recession is ideally applied to young persons with strabismus of grades 1 and 2 in which the rotations show bilateral equal excess of the internal rectus muscles associated with fairly normal external rectus muscles.

2. Recession and resection are most applicable to cases in which there is unilateral preponderance of the muscle abnormalities and for strabismus of grades 3 and 4. It is the method of choice in most cases.

3. The surgical procedure should be directed toward the muscles which are primarily at fault.

4. There are two main surgical causes for unsatisfactory results: (1) undercorrection and overcorrection and (2) failure to select the proper procedure in the given case.

5. Graduated and standard amounts of correction are indicated in the various degrees of strabismus, in the absence of reasons to the contrary.

6. The main reasons to the contrary are: (*a*) the presence of contracture, hypertrophy and atrophy of the muscles; (*b*) unusually excessive or diminished muscular action; (*c*) the presence of the various congenital defects, and (*d*) previous operations for strabismus.

7. No statistical confirmation was available which suggested that the recession-resection technic is more applicable to patients with amblyopic eyes than is bilateral recession.

MEDICAL TREATMENT OF SENILE CATARACT

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In the future more patients with senile cataract will come to the ophthalmologist for treatment. Because of the reduction of infant mortality and the lengthening span of life for those who survive the hazards of infancy, the trend of population in the industrially advanced countries is toward old age. In the United States, over one third of the total population will soon be over 50 years of age, and by 1980 over 14 per cent will be over 65. This trend in the chronologic distribution of the total population by age [1] is clearly shown in the accompanying chart. Along with this general senescence in the population belongs an increased incidence of senile cataract.

The modern treatment of senile cataract is surgical. Operation for cataract in many respects is a radical treatment after the disease has occurred. It is a removal of a part of the eye for which there is no restoration of function without a troublesome artificial substitute. In the surgical treatment of senile cataract the ophthalmologist is in the same position as the orthopedic surgeon is in correcting the osseous deformities of severe rickets. If ophthalmology constantly advances, the treatment will tend to be, first, preventive; secondly, medical, and as a last resort, surgical. Whether or not these objectives will be reached will depend on research and an enthusiastic scientific frame of mind and not on negativism.

In his excellent and remarkable article on "Further Research on Eyes of Monozygotic Twins of Advanced Age with Reference to the Heredity of Senile Symptoms," Vogt [2] asserted that the chemical studies of the lens are sure to be as futile as the chemical analysis of the optic nerve in Leber's atrophy. Furthermore, in the transactions of the German Ophthalmological Society he [3] severely criticized Müller and

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From the Division of Ophthalmology, Department of Surgery, University of Chicago.

1. Dublin, L. I.: Longevity in Retrospect and in Prospect, in Cowdry, E. V.: Problems of Ageing, Baltimore, Williams & Wilkins Company, 1939, p. 100.

2. Vogt, A.: Weitere Augenstudien an eineiigen Zwillinger höheren Alters über die Vererbung der Altersmerkmale, Klin. Monatsbl. f. Augenh. **100**:497, 1938.

3. Vogt, A.: Zur Stardiskussion in Heidelberg, Klin. Monatsbl. f. Augenh. **101**:530, 1938.

indirectly all others who are working on the treatment of senile cataract. He stated that because this type of cataract is hereditary it is useless to try to study it chemically. Those who are unfamiliar with the progress in the chemistry of genetics, senescence and the lens may agree with Vogt's point of view. This unqualified attitude is unnecessarily pessimistic in view of the existing data, for there is evidence that senescence can be influenced by various factors.

The contrary opinion is well presented by von Szily,[4] who said in a recent lecture:

> Whether senile cataract, being a hereditary characteristic of old age, will prove amenable to therapy, even if only of a prophylactic nature, remains to be proved. To reject the idea as such, in the sense of a working hypothesis, would seem, however, to be just as much out of place. Who would have imagined only a short time ago, to take just a single instance, that vitamin C, produced from the fruit of the paprica, would at any time be called upon to play such an important part just in the metabolism of the lens? New knowledge always reveals new possibilities, and it is wiser not to lay chains upon the urge for investigation in any direction whatever.

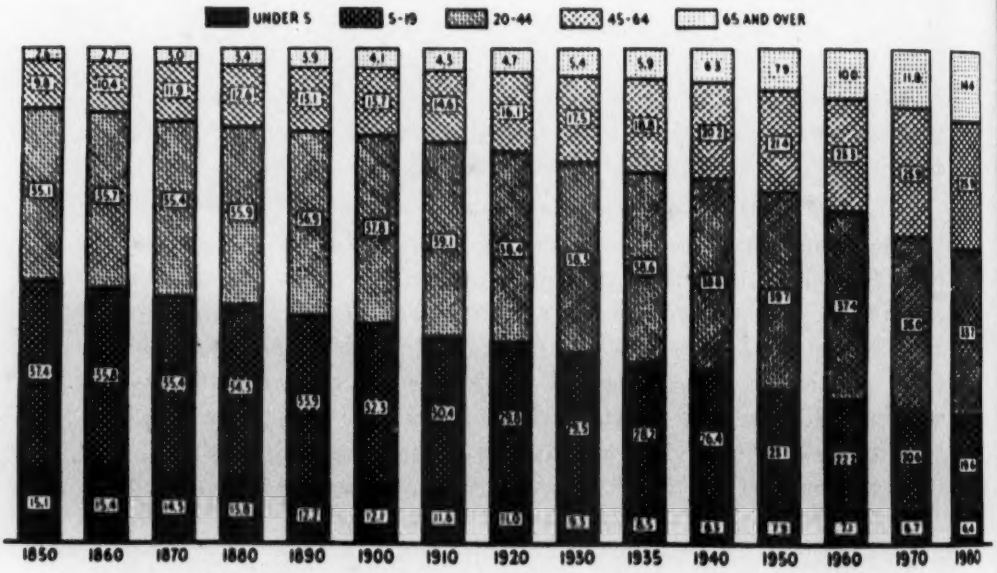

Chart showing the percentage distribution of the total population by age in the United States from 1850 to 1980. (From Dublin, L. I.: Longevity in Retrospect and in Prospect, in Cowdry, E. V.: Problems of Ageing, Baltimore, Williams & Wilkins Company, 1939, p. 100.)

4. von Szily, A.: Doyne Memorial Lecture: The Contribution of Pathological Examinations to the Elucidation of the Problems of Cataract, Tr. Ophth. Soc. U. Kingdom (pt. 2) **58**:595, 1938.

The future of medical treatment of senile cataract is not as hopeless as it may appear at the present moment. Since there is good evidence that heredity has an influence on the time factor for lenticular senescence and senile cataract, heredity, senescence and senile cataract may be considered separately. There is no doubt that heredity is a physicochemical mechanism that may be altered in the laboratory and is altered in nature. Many kinds of changes in heredity have been produced experimentally. There is no need to discuss this point in detail, for it is covered by many treatises. Although the knowledge of the experimental technic in heredity is relatively scanty, an excellent beginning has been made in the modification of heredity. Because there is no convincing experimental work which has been done on the alteration of the heredity of cataract, there is no reason why productive research cannot be done on this problem. Clinically the interest has been active in the observational stage, but experimentally it has been passive. The fact to be emphasized is that heredity is basically chemical in nature and can be changed by man.

To show that some hereditary diseases can be treated, the following examples are chosen as they come to mind, but many others, particularly inborn errors of metabolism, could be added to the list. Owing to a hereditary factor, diabetes mellitus may occur at an early age, but no physician would fail to treat the disease with insulin and a modified diet. Uniovular monozygotic twins may be born with hypothyroidism, which can be corrected with the administration of thyroxin. In xeroderma pigmentosum there is an inborn excess of porphyrins which gives a cutaneous sensitiveness to actinic light. It is fitting to prevent the exposure of this type of presenile disease of the skin to excessive light. There are, of course, hereditary diseases which are not affected by therapy. For example, choroidal coloboma cannot be changed after it occurs, no matter how much research will be done in the future. It is not logical, however, to compare such diseases with senility of the lens.

The time factor of senescence can be modified in the laboratory. MacNider, of the University of North Carolina, has produced premature senility of liver cells. Carrel, at the Rockefeller Institute, has increased the longevity of mice by sheltered life. MacKay, at Cornell University, prolonged the life of rats by restriction of the protein diet. Sherman, at Columbia University, found that calcium, vitamin A and riboflavin (vitamin G) are related to an increased length of life. Simons and Stillman, from the same university, showed that static senile cells can be induced to grow by an unidentified tissue substance. Senescence is therefore not a fixed chemical mechanism. There is good reason to believe that the average person may overcome a part of the biologic senescence by intelligent control of environment, although he

cannot completely avoid the consequences of the hereditary qualities which are born with him.

The biochemistry of the senescent lens has been little studied. Salit is probably the only one who has worked persistently on this problem. Others have been more interested in experimental cataract in young animals, which in most cases is not concerned with senile cataract A criticism cannot be made on chemical research of the aging lens which is hardly started and which will lag in progress until more is known about senescence, heredity and biochemistry. Such associated fields of research are always interdependent. In the study of senile cataract the application of chemistry and physics is needed to get beyond normal and pathologic anatomy. Physiologic and pathologic senescence are to be differentiated. The effect of infection, intoxication and trauma should be determined, for many senile cataracts may be classified as complicated cataracts. Senescence of the lens may also be a senescence of tissue, which may or may not be a part of the general senescence of the body. The question arises whether or not senile cataract is a primary result of senescence or the secondary response to alterations in the body fluids which have not maintained a proper equilibrium in the aging of the person. Of course there is no possibility of reversing the change which has occurred in a mature or hypermature senile cataract, but there is an expectation that the primary causal factor and the secondary factors which initiate or hasten the maturity of the cataract may be influenced to prevent or retard the cataractous process.

In spite of Vogt's opinion of the hopelessness of the medical treatment of senile cataract, the biochemists are interested in the growth, aging and diseases of the lens and are trying to pass beyond the pathologic changes seen in the tissues and empiric treatment. In speaking of the progress of chemical ophthalmology before the Canadian Ophthalmological Society, Duke-Elder [5] stated:

> . . . . but it is again, I think, undergoing a revolution—a revolution less spectacular and dramatic than the previous one because we are delving much more deeply into more subtle, more fundamental, and more difficult things. Hitherto we have been dealing grossly with cause and effect, and have busied ourselves with the structural ruins which disease has left behind; now we are probing more deeply to try to determine the more subtle nature of the initial defect, to pass from the study of structures composed of cells as in 1881 to the study of cells composed of molecules or atoms, to look beyond anatomy and pathology to biophysics and biochemistry.

5. Duke-Elder, S.: Progress in Ophthalmology, Canad. M. A. J. **39**:419, 1938.

BILATERAL SYMMETRIC CYSTOID DETACHMENT OF THE RETINA

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Only in the last few years have there been several reports of clinical descriptions of cysts of the retina. These cysts have usually been associated with detachment of the retina. Most of them were solitary and unilateral. Several observers have reported bilateral and symmetric cysts of the retina. Prior to these reports, which will be discussed later, most retinal cysts have been seen on pathologic examination of eyes that were removed for one reason or another, usually painful, hard eyes. The present case is of interest because it shows a bilateral symmetric cystlike detachment of the retina in a location which has not been previously observed. Also, the patient had no complaints referable to his condition, and it was seen on routine examination. It is my belief that this condition will be found more frequently if the observer is aware of its existence and if a special search is made for it in certain types of cases.

REPORT OF A CASE

A white man 33 years of age came to the office complaining of poor vision, difficulty in reading and occasional but severe headaches. His previous history was unimportant except for scarlet fever in childhood. He had been wearing glasses for the past twenty-five years, and one physician had prescribed bifocal lenses a year before. There was no history of injury to the eyes.

On examination, the palpebral fissures were seen to be rather small. They measured 26 mm. in length and 8 mm. in width. The patient had marked exophoria under cover for near vision but orthophoria for distant vision. With homatropine hydrobromide cycloplegia, a compound hyperopic astigmatism was noted. The vision in the right eye was 20/200 and was improved to 20/40 with a + 4.50 sphere combined with + 4.50 cylinder at axis 92. In the left eye the vision was 16/200 and was improved to 20/70 with a + 4.25 sphere combined with a + 5.00 cylinder at axis 95. The cornea was slightly smaller than normal. In the right eye it measured 10 mm. vertically and 10.5 horizontally. The left cornea measured 10 mm. vertically and 11 mm. horizontally. The media were clear; the tension was normal. On examination of the fundi, the disks were seen to be small and reddish, with some indistinctness of the margins. The maculas were normal. The arteries were slightly tortuous, and the veins were normal. In each eye in the extreme periphery of the lower field at 6 o'clock near the ora serrata, a spherical,

Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 20, 1939.

sharply defined elevation of the retina was noted. When looked at by indirect illumination, the outlines of the cystlike detachments could be well seen. They had a sharp margin and were distinctly spherical. The extreme lower boundary, however, was beyond the line of vision of the observer and could not be made out. The color was grayish pink, and the surface was slightly irregular and pitted, comparable to the skin of an orange. Several whitish lines were seen on the surface. When looked at by direct illumination with a + 10 sphere in the ophthalmoscope, the detachments appeared to be semitransparent, so that the choroidal vessels could be made out and the margins were much less distinct. In the left eye the termination of a small retinal vessel could be seen coming over the margin of the cyst and terminating in a whitish line. Another interesting finding in the fundus was the appearance of the retina in certain areas. There were numerous bright reflexes not usually found. This has been described by Treacher Collins[1] as a watered silk retina, observed frequently in persons with high hyperopia.

Fig. 1.—*A*, cystoid detachment of the retina of the right eye at 6 o'clock, observed by indirect illumination. This is a photograph of a colored drawing of the fundus. *B*, cystoid detachment of the retina of the left eye at 6 o'clock, observed by direct illumination. The somewhat transparent character is brought out. The choroidal vessels can be seen shining through the cyst.

The rest of the retina appeared to be normal. The fields of vision were taken, and a small defect in the upper field, corresponding to the detached area, was noted in each eye. The patient has been seen several times since the first visit on Dec. 12, 1938. At the time of writing, six months later, there had been absolutely no change in the size, shape or appearance of the cysts.

REVIEW OF LITERATURE

This unusual finding of bilateral symmetric cystoid detachment of the retina prompted a study of the literature. The most comprehensive

1. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925.

recent contribution is an article by Weve.[2] He cited 10 cases of cysts of the retina associated with detachment. The cysts are usually unilateral and located in the lower temporal field. They are always associated sooner or later with a clinically manifest detachment of the retina. Weve's hypothesis is that the cyst is always in the region of the tear of the retina and may be responsible for the tear. Case 5 of this group is that of a man of 64 with bilateral symmetric cystlike detachment of the retina in the lower temporal region. The patient had hyperopia of 1.5 diopters in each eye. The vision had suddenly become poor and for this reason the patient sought medical aid. The cysts were spherical and transparent. After rupture of the cyst in one eye by a single diathermic puncture, the retina flattened out and the vision improved from 0.1 to 0.5. The other eye was treated in the same

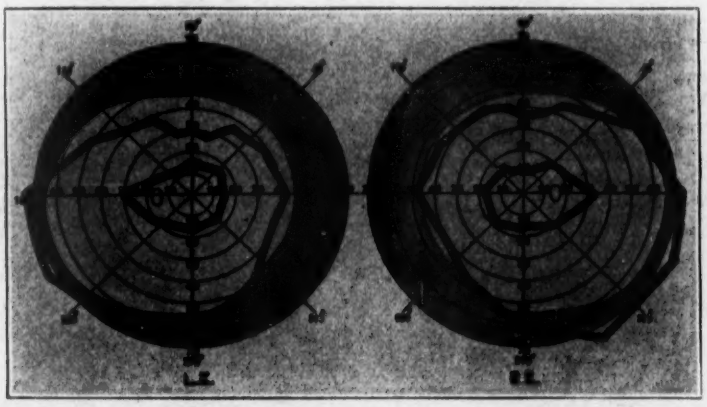

Fig. 2.—Peripheral fields of vision of the patient. Notice the small defect in the upper field. The inner black line is the field of vision for a 3 mm. red object. The form fields were taken with a 3 mm. white object at a distance of 300 mm.

manner, and there was improvement of vision from 0.5 to 1.0. Of interest in this connection is the fact that the field defects noted before operation did not improve afterward. According to Weve, this unexpected result seemed to prove the cystlike nature of the detachments. Judging from the pictures and the field defects, the cystic detachments in this case were much larger than those seen in my case.

Ridley [3] reported 5 cases of retinal cysts, in 2 of which the condition was bilateral. In 1 of these cases the patient, a man aged 64, complained of flashes of light and the presence of shadows in the visual field. On examination, retinal cysts were found in each eye in the lower temporal

2. Weve, H.: Die Beziehungen zwischen den grösseren isolierten Netzhautcysten und Netzhautablösung, Arch. f. Augenh. **109**:49, 1935.

3. Ridley, H.: Some Practical Points in the Treatment of Simple Detachment of the Retina, Brit. J. Ophth. **19**:101 (Feb.) 1935.

quadrant. In the other case the patient, a man aged 57, complained of shadows in the upper nasal field of the right eye. Examination revealed a large cyst in the lower temporal field. A little while later a cyst was noted in the lower temporal field of the left eye and a recurrence in the right. Ridley expressed the belief that the detachment was in the nuclear and reticular layers of the retina rather than between the pigment epithelium and the retina. After operation there was no improvement in the field of vision.

There are several reports in the literature of cysts of the retina which have been observed to form while the patient was under observation for

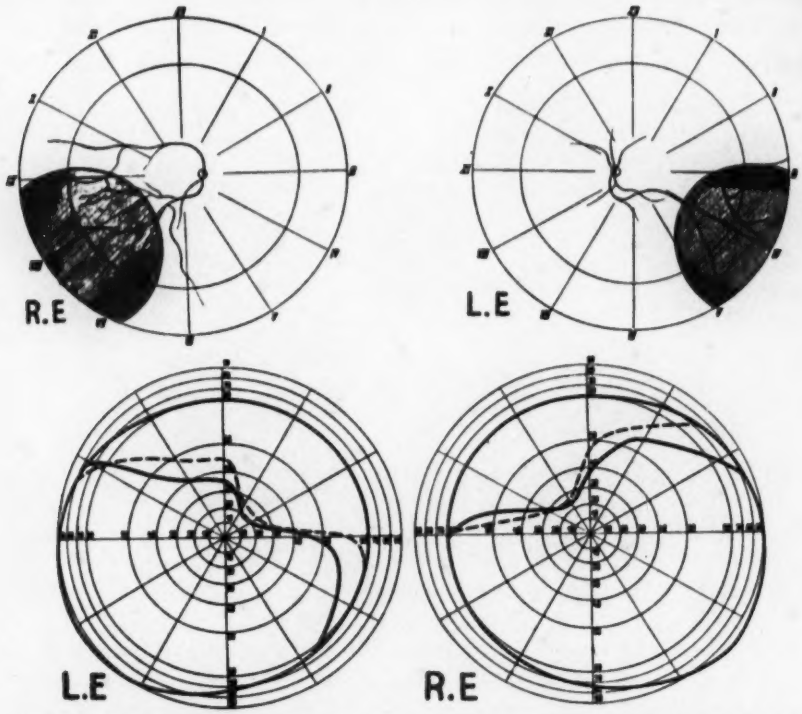

Fig. 3.—Illustrations taken from the report on cysts of the retina with detachment by H. Weve. These cysts are apparently much larger than those seen in my case. (From *Arch. f. Augenh.* **109**:49, 1935.)

some other condition. Veil and Guillaumat [4] reported a case of a young woman with an old iridocyclitis of unknown origin in the right eye which showed a bound down pupil and secondary glaucoma with cupping of the disk. While the patient was under observation a large retinal cyst developed in the left eye in the lower temporal region. There was no sign of detachment or tear. The cyst ruptured spontaneously and left

4. Veil, P., and Guillaumat, L.: Les kystes retiniens, Arch. d'opht. **2**:977 (Nov.) 1938.

as a residue a pigmented line of demarcation, showing the limits of the cyst.

Fuchs [5] reported a case in which the patient was observed by his father, E. Fuchs, from 1890 to 1925. She had chronic simple glaucoma in both eyes and was operated on. The cystic area was observed to form in the left eye in the upper outer quadrant over a period of five weeks. A similar cyst appeared temporally in the right eye eight years later. The cyst in the left eye gradually disappeared, and vision became worse because of the glaucoma. The cyst in the right eye could not be followed because it was covered by a membrane secondary to a hemorrhage. Fuchs also reported a second case, that of a man of 57, who had a cystlike detachment of the retina. It was sharply demarcated, transparent and situated in the midperiphery temporally. The retina beyond the cyst, which could be seen, appeared normal.

Van der Hoeve [6] mentioned a case of cyst of the papilla in which the cyst emptied itself into the vitreous and then refilled.

Rauh [7] reported a case of cystic detachment at the macula due to exposure to a strong glaring light. The cyst was elevated 2 diopters.

Deutschmann [8] reported a case of cyst of the retina in which the cyst was located near the ora serrata and appeared and disappeared while the patient was under observation. One eye was removed because the cystic detachment was believed to be due to a tumor. The patient had emphysema and polycythemia. Both factors favor venous stasis.

These 6 cases would appear to show that cysts of the retina may form spontaneously and rupture spontaneously.

There is a group of cases which is of interest because one could suspect the presence of a bilateral symmetric cystic formation in the retina even though the cysts themselves were not seen. This is particularly true of 2 cases reported by Schmelzer.[9] He described 2 cases in brothers, aged 25 and 32, both of whom had bilateral detachment of the lower half of the fundus with a tear at the ora serrata in the lower temporal region. There was no history of injury or birth trauma. The eyes were emmetropic or slightly hyperopic. The patients were healthy in all respects. Schmelzer agreed with Weve that a primary solitary

5. Fuchs, A.: Ueber Netzhautzysten und über die Entstehung von Netzhautlöchern, Klin. Monatsbl. f. Augenh. **98**:145 (Feb.) 1937.

6. Van der Hoeve, J.: Doyne Memorial Lecture: Eye Symptoms in Phakomatoses, Tr. Ophth. Soc. U. Kingdom **52**:380, 1932.

7. Rauh, F.: Ein eigenartiger Fall von Veränderung der Netzhautmitte, Ztschr. f. Augenh. **63**:48 (Sept.) 1927.

8. Deutschmann, F.: Ueber rezidivierende Zystenbildung in der Netzhaut, Beitr. z. Augenh. **9**:591 (April) 1914.

9. Schmelzer, H.: Doppelseitige Netzhautablösung mit symmetrischem Orariss bei zwei nicht kurzsichtigen jugendlichen Brüdern, Klin. Monatsbl. f. Augenh. **96**:19 (Jan.) 1936.

retinal cyst may have been the forerunner of the tear and detachment, the cyst being due to a maldevelopment of the retina.

Jancke [10] also reported the case of a man of 21 with a cyst in the macular region of the right eye and another cyst in the lower temporal field with a tear at the ora serrata in the same eye. There was a flat detachment in the lower temporal field of the left eye with a small tear at the ora serrata. The retina also showed a pigment ring surrounding the tear in this eye. This would seem to indicate the probable existence of a cyst in this region previous to the detachment and tear. The correction for the right eye consisted of a + 3.00 sphere combined with a + 4.00 cylinder at axis 90; that for the left eye was a + 1/50 sphere. The right eye had secondary glaucoma.

Vom Hofe [11] reported a case of symmetric bilateral retinal detachment in the lower temporal field.

This group of cases, in all of which there was bilateral symmetric retinal detachment in the lower temporal field, seems to fit in with the findings of Weve, who stated that all these detachments are due to the probable presence of a cyst in that region.

Previous to the aforementioned cases, cysts of the retina were seen invariably in eyes that had been removed for some other condition. Of especial interest in this connection is an article by Fuchs,[12] who reported 99 cases of cysts of the retina. They were all seen on pathologic section and were rarely seen clinically. The cysts were present in eyes with inflammatory disease, detachment of the retina and secondary glaucoma. He divided the cysts into 6 groups as follows: (1) cysts on the outer side of the retina between the retina and the choroid; (2) cysts on the inner side of the retina or in the retinal substance between the inner and the outer limiting membrane; (3) cysts caused by atrophy of the retina; (4) cysts associated with secondary glaucoma without detachment of the retina; (5) cysts associated with tumors of the choroid, and (6) cysts associated with detachment of the retina.

Leber [13] as far back as 1877 saw cysts of the retina on section of pathologic eyes and wondered why they were not seen ophthalmoscopically.

For the sake of completeness, I wish to mention a few other cases that have been reported in the literature of cysts of the retina or

10. Jancke, G.: Echte Netzhautzysten und Netzhautablösung beim Jugendlichen, Klin. Monatsbl. f. Augenh. **95**:145 (Aug.) 1935.

11. vom Hofe, K.: Netzhautablösung und Lebensalter, Klin. Monatsbl. f. Augenh. **93**:745 (Dec.) 1934.

12. Fuchs, A.: Zur Pathogenese und Anatomie der Netzhautzysten, Arch. f Ophth. **105**:333, 1921.

13. Leber, T.: Die Krankheiten der Netzhaut, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1934, vol. 7, pt. 2, p. 1699.

suspected cysts of the retina. Van der Hoeve [6] mentioned cysts of the retina in phakomatoses. He included in this group Bourneville's disease, the Hippel-Lindau syndrome and Recklinghausen's disease.

Meller and Marburg [14] reported a case of cyst of the retina associated with Czermak-Hippel disease of the retina.

Velhagen [15] reported 2 cases of cysts of the retina, in 1 of which the cyst could be seen with the ophthalmoscope. Both patients had glaucoma, and the eyes had to be enucleated.

Derby [16] reported a case of cyst of the retina on the temporal side of the disk. It was elevated at 6 diopters and was separate from the retinal detachment in the eye.

Gonin [17] had a different theory for the formation of cysts of the retina. In his book on detachment of the retina he has two microscopic sections showing cysts of the external surface of the detached retina. There was a tear at 5 o'clock. He stated that he did not believe that the cysts have any relation to the production of a tear of the retina as a cause. They are more likely to be an effect of the tear. In this connection it is interesting to note that cystic degeneration of the macula is a late sequel of retinal detachment.

Anderson,[18] in a discussion of anterior dialysis of the retina in 1932, mentioned 1 case in which there was a sharp crescentic line near the ora serrata. This line may indicate the presence or the previous existence of a cyst in that region. This article is interesting, because he mentioned four features which are characteristic of cases of the so-called disinsertion or anterior dialysis of the retina.

1. The condition is most commonly seen in the inferior temporal quadrant.

2. The male sex is usually affected.

3. The refraction is nonmyopic.

4. The age of onset is usually much lower than that in other cases of detachment.

Davidson,[19] in the discussion of my paper, mentioned a case of a man aged 35, who was injured in the right eye by a blow from a fist. The first ophthalmologist who saw him reported ecchymosis and hyphemia.

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14. Meller, J., and Marburg, O.: Zur Kenntnis des Wesens der sogenannten Czermak-von Hippelschen Netzhauterkrankung, Ztschr. f. Augenh. **66**:1 (Sept.) 1928.

15. Velhagen, C.: Ueber Zystenbildung in der Retina, Klin. Monatsbl. f. Augenh. **50**:716 (Dec.) 1912.

16. Derby: Retinal Cysts, in Jackson, E.: Ophthalmic Year-Book for 1911, Denver, Herrick Book & Stationery Company, 1912, vol. 9, p. 369.

17. Gonin, J.: Die Beziehungen der Retinalzysten zur Netzhautblösung, Verhandl. d. ausserord. Tag. d. ophth. Gesellsch. (1921), 1922, p. 273.

18. Anderson, R.: Anterior Dialysis of Retina: Disinsertion or Avulsion at the Ora Serrata, Brit. J. Ophth. **16**:641 and 705, 1932.

19. Davidson, M.: Personal communication to the author.

When he was seen by Dr. Davidson several months later, the pupil was found to be larger than the left, but round; a large number of pigment particles were seen in the vitreous and what looked like a giant retinal tear in the lower field near the equator. It measured about 6 disk diameters in length and 1 disk diameter at its widest, in the center. It was elliptic in form, and the edges appeared everted. At each end vessels could be seen bridging the gap. The patient has been followed for five and a half years and during this time has presented the same appearance as when first seen. The vision is 20/20 — 2. Davidson expressed the belief that there is a rupture of the retina, but the size and the stationary condition led him to suspect that there was originally a retinal cyst which had been ruptured by the contusion.

I have been particularly careful to give as comprehensive a review of the literature as I possibly could, because I feel that this type of case

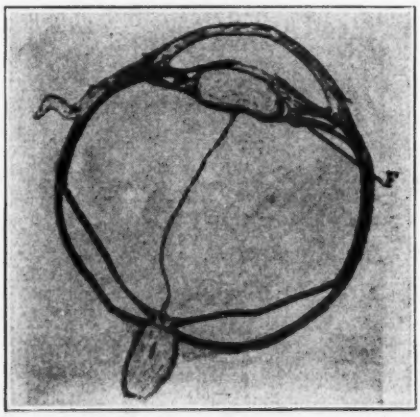

Fig. 4.—A retinal fold at the ora serrata in a microphthalmic eye. (From Ida Mann's "Developmental Abnormalities of the Eye." The illustration is published through the courtesy of the *British Journal of Ophthalmology* and the Cambridge University Press.)

forms a distinct group. It is more than a coincidence that so many of these patients with detachment of the retina in the lower temporal quadrant, occasionally bilateral, are men, usually between the ages of 20 and 40. It would seem to show that there is a group of patients, hyperopic as a rule, who have a predisposition to the formation of cysts, detachments and tears in this region.

ETIOLOGY

The question of the causation of these cysts is of great interest. Weve [2] in his report gave two possibilities:

1. The cyst may be due to an injury, however slight, which the patient does not even remember. The lower temporal location is a favorite site for contusion of the eyeball.

2. The second possibility is that it is a congenital anomaly. The lower part of the retina is phylogenetically a later developed part of the eye and hence more prone to show abnormalities. The symmetric arrangement in the cases with bilateral cysts cannot be explained on any other basis but that of a developmental abnormality.

I am inclined to favor the latter theory for the reasons given and for several other reasons. The eyes of my patient are what one would call relatively small. In such eyes the inner or retinal layer of the secondary optic vesicle is more readily thrown into folds. Folds of the retina in early embryologic life are common because of the rapid growth of the retinal layer in the first three months. As the eyeball enlarges, the retina is flattened out against the growing and expanding sclera and vitreous. However, in a small eye there might be some areas where this coaptation would not be complete. Such folds have been seen in microphthalmic eyes, and a likely place for them to form is in the region of the ora serrata, which is just before the pars planum of the pars ciliaris retinae. Ida Mann [20] has a section of a microphthalmic eye which demonstrates this fact.

Another possibility for the causation of these cysts is that they may be enlargements of the small degenerative cysts of Ivanoff, which are known to occur, especially near the ora serrata. Sometimes they get to be quite large, but rarely, if ever, are they large enough to be seen ophthalmoscopically. They usually are small and microscopic in size.

COMMENT

I made an attempt to determine whether other members of my patient's family were similarly afflicted. Two sisters and one brother were seen. Strangely enough, all these patients had peculiarities of their eyes, but none of them had any cysts. One sister had right hyperesotropia and indistinct margins of the optic disk. Another sister had a marked compound hyperopic astigmatism but no muscle imbalance. The brother had a left esotropia and amblyopia ex anopsia. All were hyperopic.

The question of treatment was then approached. Should an attempt be made to remove these cysts by diathermic puncture as Weve and Ridley have done or should they be left alone? In view of the possibility of detachment of the retina in the fture, this is a pertinent question. However, it was decided to wait and watch the patient, chiefly because nothing could be promised to him as a result of the operation, since he had no symptoms referable to his condition. If it is a cyst, what would the contents be? Naturally, this is difficult to determine. Whatever it

20. Mann, I.: The Development of the Human Eye, New York, The Macmillan Company, 1928.

is, it must be a highly transparent liquid like the aqueous because of the fact that the cysts are semitransparent.

I am sure that there must be similar cases, but because of the extreme peripheral position of this cystic area and because of the lack of complaints referable to it they are probably overlooked. However, a careful examination of all patients with marked hyperopia might reveal many more similar cases.

SUMMARY

A case of bilateral and symmetric cystoid detachment of the retina in a white man of 33 is reported. The condition was found on a routine examination of the eyes, and there were no symptoms referable to it. The patient had marked compound hyperopic astigmatism. These cysts are believed to be of congenital origin, and the relatively small eye of the patient favors their formation. Similar cases in the literature are grouped together, and the possibility of a syndrome is mentioned. This syndrome would include moderate to marked hyperopia and cystic formation in the lower field of the retina, occasionally bilateral, in young men.

NECROSCLERITIS NODOSA* ASSOCIATED WITH CHRONIC RHEUMATOID POLYARTHRITIS

REPORT OF CASE

HARRY EGGERS, M.D.
NEW YORK

In 1934 van der Hoeve [1] reported 4 cases of a new disease which he had named scleromalacia perforans. Two of the cases were his own and the other 2 belonged to Rochat, who had independently named the condition scleritis necroticans.

Scleromalacia perforans was described as a degenerative disease, accompanied by little or no inflammation and characterized by the appearance of multiple holes in the sclera. Some of the holes were covered by conjunctiva. Others exposed bare uvea to the outside. Adjacent holes often coalesced. The patients in all 4 cases were elderly. Three had advanced chronic rheumatic polyarthritis. One never had had any symptoms of arthritis. Van der Hoeve also mentioned that he had heard of the existence of 2 more cases of this disease, although he had not seen either of the patients. Both were reported as being elderly, and 1 suffered from chronic arthritis.

Apparently all 4 patients were brought to van der Hoeve after the holes had formed. He did not see the condition in its earlier stages. The oculist who referred 1 of the patients stated that one of the holes had been preceded by a yellowish subconjunctival excrescence which had looked like an abscess. When the yellowish excrescence was punctured, a kind of detritus came out.

Van der Hoeve cut off tiny fragments from the walls of some of the holes. Examination revealed "degeneration or chronic inflammation."

The condition in 1 case terminated in opaque corneas and a large hypopyon in each anterior chamber. The latter development was ascribed to infection through the bare uvea.

From St. Luke's Hospital, medical service of Dr. L. W. Frissell and ophthalmic service of Dr. W. Guernsey Frey Jr.

* The name necroscleritis nodosa was suggested by Verhoeff and King.[3] This name, or Rochat's name, scleritis necroticans, seems much more appropriate for the condition in the case here reported than does van der Hoeve's name, scleromalacia perforans.

1. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. **11**:111 (Jan.) 1934.

In the fourth case, according to the history reported to van der Hoeve, attacks of pain and redness had occurred almost once a week for thirty years. This history, the fact that van der Hoeve saw the condition in these cases in the end stages and subsequent case reports by other authors mentioning inflammatory signs all serve to cast some doubt on van der Hoeve's statement that inflammatory signs do not characterize this disease. Very likely it can occur in forms of varying severity.

In 1934 Eber [2] reported the development of a small spontaneous subconjunctival hole in the sclera, at the limbus, in a 65 year old man who had suffered from chronic arthritis since the age of 10. The hole had penetrated into the anterior chamber, thereby giving rise to a filtering subconjunctival bleb. No inflammatory signs were present when Eber saw the patient.

In 1937 Oast [3] reported the case of a man of 76. He had no evidence of arthritis. There first appeared a slightly painful, red, deeply seated nodule with a yellowish necrotic center which looked like an abscess. Incision revealed caseous material. After a few weeks the nodule absorbed and left the uvea exposed beneath the conjunctiva. Subsequent nodules appeared and repeated the same cycle. Some of the holes coalesced. Only one eye was involved. The cornea remained clear, but the vitreous became filled with opacities. All the usual laboratory tests gave negative results. Biopsy showed granulation tissue and cellular debris. Inoculation of the caseous material into the eyes of rabbits failed to show tuberculosis. Cultures for fungi and anaerobic bacteria were negative.

Kiehle [4] in 1937 reported the case of a 64 year old woman who had suffered since the age of 21 from chronic recurring deforming polyarthritis. Over a period of seven years the sclera of both eyes had gradually lost its normal whiteness and looked slate gray because a thinning of its walls allowed the choroid to show through. The episcleral tissue throughout, after a stage of edematous swelling, had been absorbed completely. When the patient was first seen the corneas and media were clear. At the time of the report both lenses were cataractous. The left cornea had become clouded. Both irides showed multiple radiating perforations. Burning and irritation, but no pain, were present.

In 1938 Verhoeff and King [5] reported a case in which enucleation was eventually done. The patient, a 52 year old man, had been

2. Eber, C. T.: Fistula at Limbus (Scleromalacia Perforans), Am. J. Ophth. **17**:921 (Oct.) 1934.

3. Oast, S. P.: Scleromalacia Perforans, Arch. Ophth. **17**:698 (April) 1937.

4. Kiehle, F. A.: Scleromalacia, Am. J. Ophth. **20**:565 (June) 1937.

5. Verhoeff, F. H., and King, M. J.: Scleromalacia Perforans, Arch. Ophth. **20**:1013 (Dec.) 1938.

afflicted with advanced rheumatoid arthritis for eleven years. When he was first seen he gave a history of redness and soreness in one eye of one year's duration and tumors of three months' duration. The eye was found to have five nodules in the anterior portion of the sclera, with congestion of the overlying conjunctiva.

When a specimen was taken for biopsy 3 drops of thick yellowish pus escaped. Smears revealed pus cells and occasional plasma cells but no bacteria or fungi. All cultures, including those for anaerobes and fungi, were negative. Inoculation of animals failed to show tuberculosis.

Eight weeks after the biopsy specimen was removed the nodule had disappeared and was replaced by a circular hole in the sclera. However, the eye became increasingly painful and congested, and since it was blind from cystoid degeneration of the macula it was enucleated at the patient's request.

Histologic examination showed the disease to be limited to the anterior part of the sclera. The scleral nodules were abscesses consisting of central masses of necrotic pus cells containing fragments of necrotic scleral tissue and surrounded by walls of epithelioid cells. Beyond the walls of epithelioid cells the scleral clefts were packed with plasma cells and fibroblasts. In places the underlying uvea also was involved. Its stroma was largely replaced by new fibrous tissue, chronic inflammatory cells and fibroblasts. The scleral hole showed partial absence of conjunctiva and episclera and replacement of the sclera and uvea by fibrous tissue containing pigment cells. New epithelium formed an external covering.

Verhoeff and King expressed the belief that the pathologic sequence is as follows: First, a small portion of the sclera becomes necrotic. It then quickly becomes walled off by epithelioid cells. Later, pus cells pass through the wall to the necrotic area, which they infiltrate and surround. This causes sequestration and disintegration of the necrotic sclera.

Several subcutaneous nodules from patients ill with rheumatoid arthritis were excised and examined by Verhoeff and King. One nodule showed a large sharply defined area of necrosis. They expressed the belief that the subcutaneous nodules of rheumatoid arthritis and the nodules of scleromalacia perforans are essentially similar in their early stages.

REPORT OF A CASE

On April 5, 1938, a 37 year old white American woman was admitted to the medical service of St. Luke's Hospital, complaining of painful swollen joints of two years' duration, associated with increasing loss of weight and weakness. Also, in the preceding three and a half months both eyeballs had been painful and congested.

Physical examination revealed severe generalized atrophy of the muscles, contractures of the flexor tendons of the arms and legs, enlargements of the meta-

carpophalangeal joints and tenderness of the shoulder, elbow and wrist joints. A diagnosis of rheumatoid arthritis was made.

The patient remained in the hospital for one hundred and sixty-six days, until she died. Emaciation became extreme. Her back and most of the joints of the extremities became unbearably painful and intolerant of any motion. Large doses of morphine failed to relieve the pain fully. The temperature ranged between 99 and 100 F.

All laboratory tests gave essentially negative results except for a constant elevation in the sedimentation rate to between 100 and 130 mm. per hour. Roentgenograms showed generalized atrophy of the articular cartilages with erosion of some of the bones. The teeth and paranasal sinuses showed no focal infection.

Therapy failed completely. Before the patient's admission to the hospital she had been treated by a competent general practitioner with diathermy and other forms of physical therapy. In the hospital she received, among other things, liver extract,

Appearance of the eyes shortly before biopsy.

ascorbic acid, calcium gluconate, synthetic crystalline vitamin B_1 (betaxin), antihistamine, typhoid vaccine intravenously and cobra venom.

Autopsy did not include examination of the head and revealed chronic atrophic arthritis with terminal acute fibrinous pericarditis and bronchopneumonia. Examination of the distal end of the right humerus showed an irregular thickening and rarefaction of the cortical bone, with a fatty gelatinous marrow. The right knee joint showed thinning of the articular cartilage with complete denudation in some areas and small hemorrhages along the margins of the cartilage. Postmortem culture of the vitreous of the eye did not produce any growth.

The eyes were examined a few days after the patient's admission. Marked bilateral conjunctival and episcleral congestion were present. Both eyes were highly myopic, with liquid vitreous and the usual myopic changes in the fundus. With her glasses, the patient could read newsprint with each eye.

Gradually, within the next three weeks, three comparatively large swellings—two in the right eye and one in the left—appeared beneath the conjunctiva and apparently in the sclera. Each swelling was composed of from one to three yellowish nodules. The swellings in each eye were limited to the upper anterior portion of the sclera and ranged in area from about one fourth to one half of the corneal area. The congestion in both the superficial and the deep blood vessels became maximal

and remained that way. The eyes were painful, but the pain in the joints was more severe and distracted the patient's attention.

One month after the patient's admission a biopsy was done on the largest of the nodules in the right eye. After the overlying conjunctiva had been incised and undermined, the outer wall of the nodule, which looked like thinned-out edematous sclera, was incised. Several drops of moderately viscid, yellowish brown liquid ran out. A culture was taken. Two small fragments of the outer wall were excised for histologic examination. The inner wall of the abscess cavity also was formed by thinned-out sclera, thus showing the nodules to be intrascleral abscesses. The culture grew a gram-positive rod—probably a contaminant. Before further identification could be made, the culture was inadvertently destroyed. The histologic report stated that the specimen was "inflammatory tissue from the sclera."

The nodule which had been incised and drained formed again. Three months after the patient's admission vision became considerably blurred due to bilateral uveitis. About a month later all vision was lost due to massive bilateral hemorrhages of the vitreous. Shortly before the patient's death sclerosing keratitis developed in the right cornea.

COMMENT

The simultaneous involvement of sclera, bones and joints in this case, and in most of the cases reported under the designation scleromalacia perforans, need not seem strange if one remembers that all three tissues are of mesodermal origin. The mystery lies in the failure to grow any organism on culture. Scleral abscesses and nodules are well known in other diseases and conditions which do not affect the articulations or bones.

Pyogenic metastatic scleritis is not an unknown occurrence. Most instances of this seem to have been caused by staphylococcic emboli which reached the sclera by way of the ciliary arteries. The lesions appear suddenly as localized, tender, acutely inflamed swellings. Abstracts of a few typical reports follow:

Terrien and Favory[6] called attention to the fact that localized suppuration of the sclera is uncommon in comparison with diffuse inflammation or tenonitis. They reported 3 cases of metastatic scleral abscesses; in 2 the abscesses were in the anterior portion of the sclera and 1 in the posterior portion. In 2 cases a staphylococcus was found. One of these patients had furunculosis. All 3 patients recovered completely. Early evacuation of the pus was recommended. The authors also referred to the classic thesis of Dollfus in 1928, in which the latter reviewed all the literature up to that time. In 90 per cent of the cases the staphylococcus was the causative organism.

Sharkovsky[7] reported a case of scleral abscess due to metastasis from an infected injury of the inguinal region. Corneal edema and iritis were present. Incision of the scleral abscess led to a cure.

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6. Terrien and Favory: Circumscribed Abscess of the Sclera, Arch. d'opht. **46**:641 (Nov.) 1929; abstracted, Arch. Ophth. **4**:112 (July) 1930.

7. Sharkovsky, I. A.: Abscess of the Sclera, Sovet. vestnik oftal. **1**:442, 1932; abstracted, Arch. Ophth. **10**:537 (Oct.) 1933.

Hudson [8] reported the occurrence of a metastatic staphylococcic abscess in the sclera at the upper limbus, developing from a carbuncle of the face. Intense iritis was present. Incision relieved the condition and revealed Staphylococcus aureus.

Hulka [9] reported the occurrence of an abscess in the upper anterior portion of the sclera accompanied by severe iridocyclitis in a patient who had just recovered from lobar pneumonia and whose blood had shown pneumococci. Incision of the scleral nodule allowed yellowish pus to run out. Culture of this showed pneumococci identical with those obtained from the patient's blood a little earlier. The eye was lost because of endophthalmitis.

Inflammatory scleral nodules are known to follow herpes ophthalmicus. Penman [10] in 1931 reviewed the literature and reported 4 cases of his own. According to Penman, the course of a typical case is as follows: Two to three months after an attack of herpes ophthalmicus, one or more dark red lentil-sized nodules appear in the sclera. The overlying conjunctiva is inflamed. Iridocyclitis is present. After several months the nodules absorb and leave sharply defined slate gray areas in the sclera. Penman's 4 patients all were elderly—over 60.

A recent editorial [11] in *The Journal of the American Medical Association* is of sufficient interest to be quoted in part. It comments on the recent discovery of an infectious polyarthritis in the wild and domestic rats of Java:

> . . . The Java pathologists were able to transfer this disease to normal rats by the plantar injection of arthritic joint exudate. . . . The infected joint cavities usually contained a cellular exudate, which was almost invariably sterile on routine bacteriologic test. Filtrates from such exudates were invariably noninfectious. . . . Afterward it was found that a fulminating type of polyarthritis could be produced in normal rats by injecting relatively large doses of arthritic exudates intraperitoneally or intrapleurally. . . . In spite of many careful attempts, it has not yet been possible to cultivate a micro-organism that might be considered to have etiologic significance. Transfer experiments using other than joint tissues obtained from sick rats indicate that the causative agent is not confined to the joints. . . . As yet no instances have been found in which complement fixing, precipitating or neutralizing antibodies have been discovered in the serum of immune rats. This disease presents exceptional features of parallelism with chronic rheumatoid arthritis in man and offers promise of yielding information of practical import.

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8. Hudson, A. C.: Staphylococcic Abscess of the Sclerotic, Arch. Ophth. **11**: 736 (April) 1934.

9. Hulka, J. S.: Metastatic Pneumococcic Uveoscleritis Following Pneumonia, Arch. Ophth. **17**:127 (Jan.) 1937.

10. Penman, G. G.: Scleritis as a Sequel of Herpes Ophthalmicus, Brit. J. Ophth. **15**:585 (Oct.) 1931.

11. Infectious Polyarthritis in Rats, editorial, J. A. M. A. **113**:148 (July 8) 1939.

THE PLACE OF COATS'S DISEASE AMONG THE DISEASES OF THE RETINA

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The place of Coats's disease, called by Coats retinitis exsudativa (retinitis externa hemorrhagica), among the diseases of the retina is still an uncertain one. The difficulty of characterizing it as a distinct entity is due to the fact that the massive exudations which are characteristic of the disease occur also in the other well characterized diseases of the retina, such as disciform macular degeneration (Junius and Kuhnt) and angiomatosis of the retina (von Hippel), and with extensive retinal hemorrhages of any origin. The difficulty is still heightened by the differences of opinion among investigators as to the cause of the exudations. Coats himself considered them to be the result of the hemorrhages in the retina. He was in doubt about the origin of the hemorrhages but leaned toward the idea that they are the result of some change in the small vessels. Other investigators, following Leber's lead,[1] considered Coats's disease to be a focal inflammatory and necrotizing process in the retina with serofibrinous exudation followed by secondary changes.

A study of the cases reported by Coats and of many of the cases reported by others has convinced me that Coats's disease is a distinct clinical entity; that the exudates and the connective tissue formation in the retina are the consequences of local circulatory disturbances there, beginning with the transudations of plasma and of blood; that the transudations and hemorrhages are the immediate results of peristasis and prestasis and of rupture of small abnormal and dilated vessels; that the small abnormal vessels represent a congenital vascular malformation involving certain branches of the central retinal vessels, and that these vascular malformations find their proper place in a classification of vascular malformations and tumors of the cerebral circulation.

COATS'S DISEASE

Under the heading "Forms of Retinal Diseases with Massive Exudation," Coats [2] described in 1908 a number of cases clinically and patho-

1. Leber, T.: Die Krankheiten der Netzhaut, in Graefe, A.; Saemisch, E. T., and von Hess, C.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelman, 1916, vol. 7, p. 1267.

2. Coats, G.: Forms of Retinal Disease with Massive Exudation, Roy. London Ophth. Hosp. Rep. **17**:440, 1907-1908.

logically and considered the original change to be in the occurrence of hemorrhages in the deeper layers of the retina. He divided the cases into three groups: (1) those in which there was no coarse vascular disease, (2) those in which marked vascular changes were present and (3) those in which there were arteriovenous communications. In the second communication [3] he excluded the last group, which were shown to be cases of angiomatosis retinae. He also entertained doubt as to whether the subdivision into the first two groups could be maintained as they showed more similarities than dissimilarities.

The essentials of the clinical picture are as follows: The disease occurs mostly in young persons and affects only one eye. In only a few cases has the other eye also become affected. The disease progresses slowly and is unnoticed until vision is greatly diminished or completely lost or a divergent strabismus calls attention to the eyes. The cornea, anterior chamber, iris and lens are normal in the early stages; the vitreous may show opacities. Characteristic of the disease is the occurrence of an exudate in the fundus, which is either single or multiple and flat or prominent and is situated in any part of the fundus. The exudate is white or yellowish white and at times gray or of a greenish color. It is situated mainly behind the retinal vessels. Hemorrhages in the retina occur in practically every case, near the veins, on the crest of the exudate or in its periphery. Glistening points and deposits of cholesterol are frequently seen. Deposits of pigment occasionally occur. The disk is usually normal and may be slightly hyperemic.

The retinal vessels present changes in a considerable number of cases, although not in all. These changes are mainly in the small vessels peripheral to the secondary and the tertiary branches and affect the veins more than the arteries. They consist of fusiform and spherical dilatations; loops, twists and tortuosities; the formation of vessels having a brushlike appearance and assuming the form of glomeruli, and of anastomosis between the vessels. The changes in the vessels are usually limited to one particular region of the retina. Coats noted small red areas near the vessels and he had difficulty in deciding whether they were small dilated vessels or hemorrhages.

The course of the disease is usually progressive. Changes occur in the exudate, with retrogression of some of the exudations and the appearance of new ones. Blood exuding from a small dilated vessel results in a large retinal hemorrhage, or it escapes into the vitreous, to be followed later by connective tissue formation (retinitis proliferans). In the later stages detachment of the retina is found; it is localized at first but later involves the whole retina. The end result is partial or

3. Coats, G.: Ueber Retinitis exsudativa (Retinitis haemorrhagica externa), Arch. f. Ophth. **81**:275, 1912.

total blindness. The end may be complicated by cataract formation, iritis, glaucoma or softening of the globe.

PATHOLOGIC CHANGES

The characteristic pathologic change is the presence of a mass of connective tissue between the choroid and the retina which may be flat or prominent. This mass is at first loose and cellular, later becoming dense. Lime granules are found here and there and when the choroid is also involved bone formation occasionally occurs. In the center of the connective tissue there is usually a space filled with a detritus consisting of broken-down red corpuscles, leukocytes, cholesterol crystals, phagocytic cells and pigment. The whole gives the picture of an exudate between the choroid and the retina which has become encapsulated with connective tissue.

In the early stages the retina is thickened with a serofibrinous exudate. In the center of the thickened area there is necrosis of retinal tissue involving the outer nuclear layer and the layer of rods and cones and occasionally involving the whole thickness of the retina. At times the serofibrinous layer extends to the inner surface of the retina, but more frequently to the outer surface. The exudate is invaded by phagocytes, the ghost cells of Coats, which according to Leber are derived from the pigment epithelium. Some of these cells are probably histiocytes derived from the adventitia of the blood vessels. In the necrotic areas are found broken-down blood elements, cholesterol and pigment. Later the necrotic areas become encapsulated by connective tissue, which gradually narrows the areas or completely obliterates them. Leber [4] pointed out that a part of this newly formed connective tissue is seemingly derived from the pigment epithelium, which shows a proliferating tendency outside of the retinal foci. Lamb [5] observed the changes from pigment epithelium to fibroblasts in cases of exudative retinitis. He found that the connective tissue thus derived was always dense and homogeneous. Internal to the encapsulating focus the retina shows cystic degeneration. Hemorrhages are found in the retina, in the subretinal space and in the vitreous. At first there are no adhesions between the choroid and the retina; however, in the later stages adhesions as well as slight cellular infiltration of the choroid are observed. The lamina vitrea is usually intact. Detachment of the retina is found, with many phagocytes and with coagulated fluid in the subretinal space.

Changes in the vessels were not found in all cases. They are as follows: (1) marked dilatation and congestion, the dilatations being so

4. Leber,[1] p. 1302.

5. Lamb, H. D.: The Pathogenesis of Some Intraocular Osseous Tissue: True Metaplasia in the Eye, Am. J. Ophth. **18**:409, 1935.

marked in some cases as to give the appearance of a cavernous angioma; (2) thrombi in various stages of organization in some of the dilated vessels; (3) thickening and hyaline degeneration of the vessel walls; (4) perivascular accumulation of cells in a few cases, and (5) proliferation of the endothelium and loss of staining of their nuclei in a few cases.

ETIOLOGY

Coats's presentation of his clinical and pathologic material and his pathogenic deductions are a masterpiece of clear thinking and exposition. Coats came to the conclusion that the pathologic process is initiated by hemorrhages into the retina derived from the capillaries in the external reticular layer. The extravasated blood either pushes the external limiting membrane outward or causes a rupture and forms a localized or diffuse extravasation. This extravasation of blood is responsible for the destruction of the retinal elements, for the serofibrinous fluid in the neighboring parts of the retina and for the cystic degeneration of the retina. The extravasated blood causes a reactive influx of phagocytes and a proliferation of connective tissue, which encapsulates it. The central part gradually becomes liquefied and is filled with debris of cells and cholesterol. The edema in the adjacent parts of the retina extends into the subretinal space, with resulting detachment of the retina. The reactive process, which resembles a mild inflammatory process, occasionally extends to the choroid, with the formation of adhesions between the retina and the choroid. Coats found no evidence of an inflammatory process and did not consider any such process as an etiologic factor.

The question arose as to what caused the hemorrhages. In an occasional case Coats considered hemorrhage due to a forcible forceps delivery or to a sudden increase in intravascular pressure as a result of whooping cough. However, in the greater number of cases such a causation could not be assumed. Coats considered therefore two possibilities: (1) an abnormal constitution of the blood, which is responsible for the hemorrhage, and (2) a primary change in the vessels, especially the smaller ones. He inclined toward the second of the two possibilities, although in some cases no vascular changes were found. He considered dilatation of the vessels and their increase in number a secondary change.

The clear insight of Coats into this disease incites admiration. It is unfortunate that Leber [1] did not agree with Coats's interpretation of the pathogenesis of the disease and assumed an inflammatory and necrotizing process in the retina as the cause. A number of authors have followed him in this interpretation, although a critical examination of the pathologic changes and of the course of the disease is strictly against it.

A study of the pathologic reports of Coats's disease shows that in spite of the necrosis of the retinal elements, which would argue for a severe inflammation, some of the essential elements of an inflammatory process in the retina are not present. There is no outpouring of leukocytes into the retina and no clouding of the vitreous with inflammatory elements; there is only a mild involvement of the choroid by round cells, and then only in some cases, evidently of a secondary character. The notion that Coats's disease is an inflammatory process of the retina cannot be entertained by Leber's own criteria.[6]

The essential features of the pathologic process of Coats's disease aside from the changes in the vessels are: (1) the outpouring of plasma into the retina; (2) the hemorrhages; (3) the necrosis of the retinal elements; (4) the deposit of fibrin and lipoids among the retinal element; (5) the invasion of the retina by phagocytes, and (6) the organization and encapsulation of the hemorrhages and transudates which are too large to be absorbed. The first and second features represent the immediate result of local circulatory disturbances, while the necrosis is the result of the hemorrhages. Fibrin is deposited by the transuded blood plasma, and the appearance of lipoids is due to the disintegration of blood of necrotic retinal tissue. Invasion by phagocytes and the organization and encapsulation of hemorrhages too large to be absorbed are the further results of local circulatory disturbances. All these pathologic features together represent a good example of the results of local circulatory disturbances, such as I have outlined on another occasion.[7] The progressive course of the disease and the presence in the retina of lesions in various stages of organization make it necessary to assume that the transudation of plasma and the hemorrhage keep on recurring. The problem involved is as to the character of the vascular changes which permits repeated transudations of plasma and of blood.

IMPORTANCE OF THE DILATED SMALL VESSELS IN THE AFFECTED AREA OF THE RETINA

In discussing local circulatory disturbances [7] it was mentioned that transudation of plasma and hemorrhages occur whenever there is a certain definite slowing of the blood stream in the terminal vascular units. The slowing occurs whenever there is a constriction of the artery above the dilated terminal units, and the degree of slowing results in peristasis characterized by transudation of plasma or by prestasis characterized by the passage of blood through the capillary walls. To produce repeated transudations of plasma and repeated hemorrhages such as are found in

6. Leber,[1] p. 567.

7. Elwyn, H.: Circulatory Disturbances in the Retina in Arteriosclerosis and Essential Hypertension, Arch. Ophth. **21**:775 (May) 1939.

the retina in Coats's disease, the small terminal units, the prearterioles, the capillaries and the venules, must be repeatedly in a state of dilatation either of functional or of organic origin. There is nothing known of any functional constriction of arteries in this disease, nor does it seem possible that any functional constriction of the arteries with dilatation of the terminal units could produce the ophthalmoscopic and pathologic picture of Coats's disease. There is, however, plenty of evidence of an organic dilatation of small vessels in certain areas in the retina in this disease.

In the various pathologic reports of cases of Coats's disease the vascular change most frequently mentioned is dilatation of the small vessels in the affected area of the retina. It is found in most of the reported cases. Coats mentioned it in seven of nine pathologic reports. In case 8 the dilated vessels almost took on the appearance of a cavernous angioma; in the other cases the dilatation of the vessels was not so marked. Berg [8] reported 4 cases in which there were marked vascular changes, narrowing and dilatation of vessels, thrombi and dissecting aneurysms. His report is accompanied with unusually good colored illustrations depicting the dilatations and aneurysms. Rados [9] reported a case in which dilated vessels were found. Miyashita and Nisyake [10] reported a case in which the vessels had almost the appearance of a cavernous angioma. There was aneurysmal dilatation of the small vessels, and all the capillaries in the neighborhood were enlarged. In Sattler's [11] 2 cases the vessels were markedly dilated, as they were also in Wölfflin's [12] case, in ten Doesschate's [13] 2 cases, in Zinsser's [14] 5 cases, in Marchesani's [15] case and in von Hippel's [16] 2 cases (cases 2

8. Berg, F.: Beitrag zur pathologischen Anatomie der Retinitis exsudativa, Arch. f. Ophth. **98**:211, 1918-1919.

9. Rados, A.: Ueber die Veränderungen im Frühstadium der Retinitis exsudativa, Arch. f. Ophth. **105**:973, 1921.

10. Miyashita, S., and Nisyake, Y.: The Pathological Anatomy of Retinal Degeneration with Multiple Aneurysms, Brit. J. Ophth. **5**:448, 1921.

11. Sattler, H.: Ueber die pathologish-anatomischen Veränderungen der Retinitis exsudativa (Coats), Klin. Monatsbl. f. Augenh. **74**:222, 1925.

12. Wölfflin, E.: Beitrag zur pathologischen Anatomie der Retinitis exsudativa externa, Arch. f. Ophth. **117**:33, 1926.

13. ten Doesschate, G.: Ueber Retinitis exsudativa externa, Klin. Monatsbl. f. Augenh. **79**:505, 1929.

14. Zinsser, F.: Beitrag zur Kenntnis der exsudativen Netzhauterkrankungen (Fünf Fälle von Retinitis exsudativa, ein Fall von Angiomatosis retinae), Arch. f. Ophth. **121**:686, 1929.

15. Marchesani, O.: Zur Anatomie der Angiomatosis retinae und Retinitis exsudativa, Arch. f. Augenh. **103**:643, 1930.

16. von Hippel, E.: Angiomatosis Retinae und Retinitis Exsudativa Coats, Arch. f. Ophth. **127**:27, 1931.

and 3). Lamb[17] reported 8 cases of exudative retinitis. Dilatation of some vessels or the presence of numerous vessels is merely mentioned in his second, third, fifth and seventh cases. He considered the dilatation of the vessels as secondary and the primary condition as inflammatory.

In spite of the few cases in which no dilatation of vessels were reported, it is obvious that dilatation of the vessel and an increase in their number in the affected area of the retina constitutes one of the essential elements in the pathologic picture of Coats's disease. In such dilated vessels a slowing of the blood stream must occur and with it the states of peristasis and prestasis with the accompaniment of transudation of plasma and of blood. In addition, there is rupture of small dilated vessels, as shown by the presence of small dissecting aneurysms in some of the cases. The transudation of plasma and of blood initiates the changes in the retina which altogether constitute Coats's disease.

The numerous small vessels in the affected area of the retina, the aneurysmal dilatation, the dissecting aneurysms and the hyalinized or thickened walls evidently represent vascular malformations or angiomas localized in a certain vascular area of the retina.

In the same year and in the same volume of the *Archiv für Ophthalmologie* in which Coats's second publication[3] appeared there was also a contribution by Leber[18] on a form of retinal degeneration characterized by the presence of multiple aneurysms. Leber had 2 cases of his own and had collected 11 more from the literature. He considered these cases as belonging to a group which is partly overlapped by the second group of Coats's cases, the one in which there were marked vascular changes. One of Leber's collected cases is included among Coats's series.

An advance in the understanding of Coats's disease was brought about by the publications of Junius.[19] Junius attempted to correlate Coats's disease and Leber's retinal degeneration with miliary aneurysms with a disease known as "hereditary familial telangiectasia," also known as Osler's disease. In this disease, which has been shown to be familial and hereditary, there occur numerous telangiectases in various parts of the body. Areas of predilection are the skin and the mucous membranes, especially of the nose, with frequent occurrence of epistaxis. Telangiectases occur also in the mucous membrane of the respiratory tract and the digestive and the urogenital tracts as well as in the brain and in the eyes. These telangiectases consist of dilated capillaries and

17. Lamb, H. D.: Exudative Retinitis, Am. J. Ophth. **21**:618, 1938.

18. Leber, T.: Ueber eine durch Vorkommen multipler Miliaraneurysmen charakterisierte Form von Retinaldegeration, Arch. f. Ophth. **81**:1, 1912.

19. Junius, P.: Zur Aetiologie der Retinitis exsudativa Coats, Klin. Monatsb'. f. Augenh. **92**:748, 1934; Angiomatosis retinae—Retinitis exsudativa Coats—Morbus Osler, Ztschr. f. Augenh. **84**:193, 1934.

venules which have a tendency to bleed. The primary disturbance is considered by some investigators to be a defect of the mesenchymal tissue, which fails to develop normal endothelial cells lining the vessels and normal supporting tissue. The condition must be regarded as a system disease. The tendency to bleed is considered a result of the defective endothelial cells. Junius considered the possibility that the miliary aneurysms in the retina might be equivalents of such telangiectases and that they possibly represent a localized weakness in mesenchymal differentiation. The miliary aneurysms in the retina which are associated with retinal degeneration unquestionably belong to the classification of Coats's disease.

The foregoing review leads to the following logical conclusion: The fundamental pathologic factor in Coats's disease is a vascular malformation involving a certain definite area of the retinal circulation. The malformation involves the small vessels, small arteries, capillaries and veins, with the formation of miliary aneurysms, dilatation of the vessels and with defective walls which are subject to hyalinization, thickening or thinning and to rupture. Such malformations must necessarily be of congenital origin. With the years the vessels are likely to dilate more and to rupture, with clotting of the blood. The slowing of the blood flow in these vessels and the occasional ruptures are responsible for the transudation of plasma and for the hemorrhages. The transuded plasma and the hemorrhages in the retina incite the reactive changes, the invasion by phagocytes and the production of connective tissue.

The assumption that a vascular malformation which manifests itself in dilated vessels is the essential factor in Coats's disease leads to the problem whether such a malformation finds a definite place in a classificatory scheme of vascular anomalies. The retinal circulation is a part of the cerebral circulation, and it is therefore of interest to know whether the various vascular malformations in the brain find their counterpart in the retina.

CLASSIFICATION OF VASCULAR MALFORMATIONS AND TUMORS IN THE BRAIN

The classification of vascular malformations and tumors in the brain varies with different investigators. Cushing and Bailey [20] divided tumors of the blood vessels into two groups: (1) angiomatous malformations and (2) angioblastomas or true neoplasms of blood vessel elements. Bergstrand, Olivecrona and Tönnis [21] recently published a most comprehen-

20. Cushing, H., and Bailey, P.: Tumors Arising from the Bloodvessels of the Brain, Springfield, Ill., Charles C. Thomas, Publisher, 1928.

21. Bergstrand, H.; Olivecrona, H., and Tönnis, W.: Gefässmissbildungen und Gefässgeschwülste des Gehirns, Leipzig, Georg Thieme, 1936, chap. 2, pp. 8-68.

sive account of the pathologic picture of the malformations and tumors of the vessels of the brain. They pointed out that it is impossible to draw a sharp line of distinction between the two. All angiomas are primarily malformations, even when they appear as true tumors, such as the Lindau tumors. Bergstrand expressed the opinion that Virchow's classification of angiomas is still the best, and he added to it the forms of angioma which have been discovered since the publication of Virchow's lectures. Virchow [22] classified angiomas into two types: (1) angioma cavernosum and (2) angioma racemosum. The first tumor is characterized by blood spaces, between the walls of which there is no foreign tissue. In the second type each vessel forms a separate entity, and the tumor consists of a mass of vessels which are separated from one another by some other tissue. Tumors of this type (angioma racemosum) are divided into four subgroups: (*a*) telangiectasia, in which the vessels are undoubtedly capillaries but are dilated, with changes in their walls and an increase in their number; (*b*) angioma racemosum arteriale, in which the arterial branches of a certain definite vascular area are affected; (*c*) angioma racemosum venosum, in which a group of venous branches make up the vascular tumor, and (*d*) aneurysma arteriovenosum, dilated arteries and veins, in which there is a direct connection between the two. Bergstrand and his co-workers added another subgroup in which the vascular malformations resemble telangiectasia and involve in the complete syndrome the brain, the choroid of the eye and the skin. They named the syndrome Sturge-Weber disease.

To this classification Bergstrand, Olivecrona and Tönnis added two groups which were not known at the time of Virchow: (1) angioblastoma or angioreticuloma or Lindau's tumor and (2) angioglioma.

In summary, this classification of angiomas of the brain as outlined by Bergstrand and his co-workers follows:

1. Angioma cavernosum
2. Angioma racemosum
   - (*a*) Telangiectasia
   - (*b*) Sturge-Weber disease
   - (*c*) Angioma racemosum arteriale
   - (*d*) Angioma racemosum venosum
   - (*e*) Aneurysma arteriovenosum
3. Angioblastoma, angioreticuloma or Lindau's tumor
4. Angioglioma

This classification gives an adequate account of the vascular malformations and tumors which occur in the brain. I shall discuss briefly

22. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1867, vol. 3, p. 460.

the individual forms so far as they also occur in the retina. For didactic purposes, I shall take them up in the reverse order.

Angioglioma.—The term angioglioma was used, according to Bergstrand, by Roussy and Oberling for a tumor which supposedly consists of a combintaion of malformations of vessels and of nerve tissue. However, the tumor of Roussy and Oberling and similar tumors which Bergstrand examined seemed to him to belong to the group of Lindau tumors. Although these tumors do not belong to a special group, he proposed to retain the term angioglioma for tumors consisting of a combination of vessels and nerve tissue. He considered as the best example of such a tumor the subependymal tumor found in cases of tuberous sclerosis (Bourneville's disease). These consist "on the one hand of large giant astrocytes rich in protoplasm and of piloid astrocytes and on the other hand of a convolute of vessels which for the greater part are calcified."[23] In this monograph[21] a photographic reproduction is presented of an angioglioma in a case of tuberous sclerosis.

In cases of tuberous sclerosis small mulberry-shaped tumors have been found in the retina; these were first described by van der Hoeve.[24] Pathologic examination showed them to consist of nerve fibers and large cells, but the presence of any dilated vessels in them has not been reported. So while angiogliomatous tumors occur in the subependymal regions of the brain in tuberous sclerosis, they have not been reported in the retina in association with this disease.

A tumor consisting of a combination of glial elements and of miliary angiomas is described by Bergmeister[25] in a case of microphthalmos with orbital cyst. He characterized the tumor as angioglioma and mentioned a similar case reported by Seefelder.

Angioblastoma or Angioreticuloma.—This type of tumor is also known as Lindau's tumor or as Lindau's disease. When in addition to the involvement of the brain a similar tumor is also found in the retina, the condition is known as Hippel-Lindau disease. In 1926 Lindau[26] described these tumors in cerebellar cysts. The tumor has autonomous growth and is of meningoencephalic origin. It consists of angioblasts which form masses of capillaries surrounded by a network of reticulin. Between the capillaries there are endothelial cells filied

23. Bergstrand, Olivecrona and Tönnis,[21] p. 60.

24. van der Hoeve, J.: Augengeschwülste bei der tuberösen Hirnsklerose (Bourneville), Arch. f. Ophth. **105**:880, 1921.

25. Bergmeister, R.: Ueber gliöse Wuchenungen im Auge by Mikrophthalmus congenitus und deren Beziehungen zur Angiomatosis retinae, Arch. f. Ophth. **105**: 1, 1921.

26. Lindau, A.: Studien über Kleinhirnzysten, Acta path. et microbiol. Scandinav., 1926, supp. 1, p. 1.

with fat droplets resembling xanthomatous cells. The tumors occur especially in the cerebellum and also in the spinal cord and in the retina. With such a tumor, which frequently forms cystic spaces, there are also found multiple cysts in the pancreas and less often cysts in the kidneys, hypernephromas and tumors of the epididymis.

In the retina the disease is well known to ophthalmologists as angiomatosis retinae, or Hippel's disease. Here the tumor is a capillary angioblastoma, made up of angioblasts similar to those found in the cerebellum. These cells form capillary spaces and solid cellular cords. They also form cystic spaces and frequently give rise to a secondary proliferation of glia. Transudation of plasma in the retina is a frequent occurrence. Detachment of the retina occurs later.

In the early stages, before complications have set in, the ophthalmoscopic picture is striking. A pair of dilated, enlarged and tortuous vessels, an artery and a vein, course toward a raised roundish tumor in the periphery, which they enter. Several such tumors, each with a pair of dilated vessels, may be seen. Later the picture is complicated by exudates, detachment of the retina, iridocyclitis or glaucoma.

Lindau expressed the opinion that a vascular malformation in the roof of the fourth ventricle is the plausible matrix of the tumor in the cerebellum. The vascular plate is there formed at about the third embryonal month, at the same time that the retina becomes vascularized.

Arteriovenous Aneurysm.—This is a vascular anomaly in which there is an enlargement and dilatation of an artery and the corresponding vein. Both vessels are either directly united by means of one good-sized vessel or of several such vessels, or a bunch of intertwined dilated vessels are interpolated between the artery and the vein instead of the normal capillary bed. Bergstrand made the statement that the vessels which make up the arteriovenous aneurysm are so malformed as to scarcely deserve the name of either artery or vein. The structure of the walls of the vessels resembles that which makes up the walls of the vessels of the angioma racemosum venosum. Numerous cases of arteriovenous aneurysm occurring in the brain have been reported. The literature is found in the publications of Bergstrand and his associates,[21] Cushing and Bailey,[20] Dandy [27] and Brock and Dyke.[28]

In the retina arteriovenous aneurysm is a rare disease. Leber [29] mentioned 4 cases from the literature. Stokes,[30] who reported a case

27. Dandy, W. E.: Arteriovenous Aneurysm of the Brain, Arch. Surg. **17**: 190 (Aug.) 1928.

28. Brock, S., and Dyke, C. G.: Venous and Arteriovenous Angiomas of the Brain, Bull. Neurol. Inst. New York **2**:247, 1932.

29. Leber,[1] p. 37.

30. Stokes, W. H.: Racemose Arteriovenous Aneurysm of the Retina (Aneurysma Racemosum Arteriovenosum Retinae), Arch. Ophth. **11**:956 (June) 1934.

of his own, found 2 more cases in the literature. The ophthalmoscopic picture is striking. The involved vessels are dilated, enlarged and tortuous, but the arteries and veins can be distinguished and the places of anastomosis can frequently be seen. The disease is perhaps not so rare as one would judge from the few reported cases. At the meeting in May 1939 of the Section of Ophthalmology of the New York Academy of Medicine there was presented a case of arteriovenous aneurysm of the retina. Another case was seen at the New York Eye and Ear Infirmary, and excellent colored photographs were obtained by Dr. D. Bogart.

Angioma Racemosum Venosum.—The venous angioma consists of dilated venous channels. There is a gradual transition between such an angioma and a varix. The growth occurs in the meninges of the brain or in addition also in the brain tissue below the meningeal involvement.[31] The walls of the vessels involved in the angioma are markedly deformed (Bergstrand, Olivecrona and Tönnis).

In the retina dilated, enlarged and tortuous vessels occasionally occur. They have been described by Leber [32] under the heading of congenital tortuosity of retinal vessels. He reproduced in his publication a picture made in one of Nettleship's cases. Dilatation of the retinal veins is also found occasionally in what is now known as Sturge-Weber disease in combination with vascular nevi of the skin. Case 3 of Brock and Dyke is of special interest. This was a case of venous angioma of the brain in which the right eye showed on ophthalmoscopic examination that "the veins were so greatly engorged and tortuous that the disc was almost completely obscured."[28] The condition was regarded as a varicosity or venous angioma of the central vein. The angiomatous lesion extended "from the right retina and optic nerve, chiasm, and tract to the right midbrain." The tumor was composed of thin-walled blood vessels.

Angioma Racemosum Arteriale.—Arterial angioma supposedly consists of dilated arteries, the vessels leading to the tumor and draining the blood from the tumor being arterial in character. Bergstrand stated that he considered it doubtful whether such tumors actually occur, and with the walls of the vessels of racemose angioma malformed it would be difficult to characterize them as arteries. Brock and Dyke [28] classified their case 8 as one of arteriovenous angioma, although they appended to the histologic description the diagnosis, arterial angioma, made by Orton and Wolf.

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31. Bailey, P.: Intracranial Tumors, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 194. Cushing and Bailey.[20] Brock and Dyke.[28]

32. Leber,[1] p. 53.

Leber [33] mentioned under congenital tortuosity that occasionally the arteries of the retina are also tortuous with the veins. He did not mention any case in which the arteries alone are involved.

Sturge-Weber Disease.—In 1879 the English physician Sturge described a case of congenital glaucoma and naevus flammeus of one side with epileptiform convulsions of the other side of the body. He concluded that the brain must be the seat of a vascular nevus. Vascular nevi of the skin with glaucoma had already been observed before Sturge's report. In 1922 F. Parkes Weber found roentgenographically characteristic parallel lines in the brain in such a case. Since then this syndrome has attracted more attention, and Bergstrand and his associates have proposed the name Sturge-Weber disease for it.[34] In the complete syndrome there is found clinically vascular nevi of the skin, glaucoma, convulsions and frequently paresis and idiocy. However, the complete syndrome is not always present, and convulsions and vascular nevi may exist without glaucoma; or there may be vascular nevi and glaucoma without involvement of the brain, or convulsions may occur alone.

The vascular nevi may be found in the region of the distribution of the trigeminal nerve in the face, or they may occur in other regions of the skin. The disease is congenital and probably hereditary. Bergstrand and his associates have collected 108 cases from the literature in which there was involvement of the brain, among them, 5 cases of their own. In Bailey's book on intracranial tumors [21] cases 19 and 20 are such cases, apparently without glaucoma.

In the brain the characteristic lesion is a calcification of small capillaries in the cortex with a multiplication and dilatation of the meningeal vessels above the cortical lesion. The dilated vessels are veins, but the walls are very thin or are thin on one side and thickened on the other side. Leiomyomatous nodules and nodular hypertrophy of the intima occur in some regions according to Bailey.

In the eye the characteristic change on which the glaucoma seems to depend is an angioma of the choroid which is of the cavernous type. A number of pathologic reports are on record. The literature is reviewed by Granström [35] and by Bergstrand and his co-workers.[36] The cavernous angioma of the choroid is not localized, but the whole choroid becomes gradually thickened from before backward and forms a disklike tumor at the posterior pole.

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33. Leber,[1] p. 54.

34. Bergstrand, Olivecrona and Tönnis,[21] p. 20.

35. Granström, K. O.: Naevus Flammeus Associated with Glaucoma, Acta ophth. **13**:115, 1935.

36. Bergstrand, Olivecrona and Tönnis,[21] p. 33.

In the retina there need not be any vascular anomaly. However, in some of the reported cases vascular changes have been found there, consisting of dilatation and tortuosity of veins. Leber [37] mentioned the first reported case of glaucoma with vascular nevi of the skin, that of Schirmer, in which the retinal veins were dilated and tortuous, the arteries being normal. He mentioned also a case of Pantaenius, evidently with the complete Sturge-Weber syndrome, with dilatation of the retinal veins, and a case of Horrock in which there was similar involvement of the retinal veins. Mehney [38] reported a case in which there were markedly tortuous and anomalous veins in the retina, the arteries being normal.

Classification of Vascular Malformations and Tumors as Occurring in the Brain and in the Retina

Condition	Involvement of Brain	Involvement of Retina
Angioma cavernosum	Occurs but rarely	Probably does not occur
Angioma racemosum Telangiectasia	Occurs in all parts, especially in the pons	Occurs as the fundamental element in Coats's disease
Sturge-Weber disease	Consists of: 1. Calcification of the capillaries in the cortex 2. Dilatation of the meningeal vessels above the cortical lesion	Consists of dilatation and tortuosity of retinal veins (cavernous angioma in choroid); not in all cases
Angioma racemosum arteriale	Of rare occurrence	Occurs as dilatation and tortuosity of arteries in association with similar involvement of veins
Angioma racemosum venosum	Occurs in the meninges or in the meninges and the brain tissue below	Occurs as dilatation and tortuosity of veins alone or as part of cerebral involvement
Angioma racemosum arteriovenosum	Occurs in the brain	A rare disease in the retina; reported cases are small in number
Angioblastoma or angioreticuloma or angiomatosis of the central nervous system (Hippel-Lindau disease)	Occurs as cystic tumors of the cerebellum	Occurs as capillary angioblastomas, single or multiple, with a pair of dilated enlarged and tortuous vessels running to each tumor (Hippel's disease)
Angioglioma	Occurs as subependymal tumors in tuberous sclerosis	Occurs in extremely rare cases of microphthalmos with orbital cyst

Telangiectasia.—Telangiectasia differs from cavernous angioma in that there is some other tissue between the dilated vessels in the telangiectatic area. With the increase in dilatation of the vessels, the tissue between them is compressed and becomes atrophied. The vessels then resemble a cavernous angioma, and there is probably a gradual transition between the two forms of angioma. In the central nervous system telangiectasia occurs especially in the pons but also in other parts of the brain and spinal cord.

37. Leber,[1] p. 56.

38. Mehney, G. H.: Naevus Flammeus Associated with Glaucoma: Report of a Case, Arch. Ophth. **17**:1018 (June) 1937.

In the retina telangiectasia occurs not as a part of the involvement of the brain but independently. Just as it may involve a particular vascular area of the central nervous system, so it may involve a particular vascular area of the retina. When this occurs, telangiectasia constitutes the fundamental pathologic lesion which initiates the pathologic and ophthalmoscopic changes which characterize exudative retinitis, or Coats's disease.

Angioma Cavernosum.—Cavernous angioma is a rare occurrence in the central nervous system. It does not seem to occur in the retina. The dilated blood spaces which almost resemble a cavernous angioma, such as in case 8 of Coats, are probably areas of telangiectasia in which the individual vessels have gradually become more and more dilated and have compressed the retinal elements between them.

The accompanying table gives a summary of the individual forms of angioma as they occur in the brain and in the retina.

CONCLUSION

1. Coat's disease, or exudative retinitis, is a distinct clinical and pathologic entity.

2. The fundamental pathologic element in Coats's disease is a vascular malformation involving a certain definite vascular area of the retina. This malformation corresponds to telangiectasia and involves the small vessels, the terminal units, with the formation of miliary aneurysms and dilatation of capillaries and veins with defective walls, which are subject to rupture.

3. As a result of slowing of the blood stream in the dilated vessels and of rupture of these vessels, there is a transudation of plasma into retinal tissue and the occurrence of hemorrhages there. The hemorrhages cause necrosis of retinal tissue, and the plasma and blood spread and extend externally to the retina. From the plasma, fibrin is deposited in the retinal tissue.

4. The transuded plasma and the hemorrhages incite reactive phenomena: the appearance of phagocytes derived from the cells of the layer of pigment epithelium and from histiocytes of the adventitia of vessels; the formation of fibroblasts from mesodermal elements and by metaplasia of cells from the pigment epithelium layer. The fibroblasts invade the hemorrhagic areas and cause an organization or encapsulation of these areas. When the hemorrhage is encapsulated, the central part is liquefied and contains remnants of blood elements and deposits of cholesterol.

5. Telangiectasia finds its place in a classificatory scheme of vascular malformations which involve the retina in analogy to those which involve the vessels of the brain.

PLASTIC REPAIR OF CONJUNCTIVAL DEFECTS WITH FETAL MEMBRANES

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Conjunctiva has to be replaced when a large enough area is destroyed so as to cause a high degree of symblepharon or entropion or when the socket is too small to carry a prosthesis. Most common causes of its destruction are chemical and thermic burns and trachoma. Rare causes are malignant tumor, pemphigus, tuberculous ulcer, mechanical injury and amyloid and hyaline degeneration.

Conjunctiva can be replaced primarily immediately after its loss. This has to be done after the removal of the necrotic conjunctiva. Denig [1] introduced the method by which necrotic conjunctiva is removed immediately after the chemical burn and the defect is covered with mucous membrane. However, the conjunctiva usually has to be replaced when symblepharon has already developed.

Veasey [1a] revised the materials which are or have been used to cover the defects of the conjunctiva. These consist of grafts of conjunctiva, of the inner surface of the lips and of the conjunctiva of the rabbit and Thiersch grafts of the skin.

The ideal material for replacing conjunctiva is conjunctiva itself, but only a small piece can be taken from the same patient's other eye. For the restoration of a complete lower lid, Blaskovics [2] used one part of the upper fornix and adjoining tarsal conjunctiva, the corresponding strip of tarsus of the other eye being employed to build the inner layer of the lid. Some patients would refuse operation on the other normal eye in favor of the defective lid. Another material, the skin, is stiff, thick, striking in color and desquamating, and hair may grow on it. However, a sufficiently large graft is always obtainable, and therefore it is the only material for use in the restoration of the socket. The mucous membrane of the mouth is smooth and shiny and is thus a good material from the standpoint of the surface. Its

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From the State Hospital, Budapest, Hungary, Prof. Dr. J. Imre, Director.

1. Denig, R.: Early Surgical Treatment of Burns of the Conjunctiva, Am. J. Ophth. 3:256, 1920.

1a. Veasey, C. A., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

2. von Blaskovics, L., and Kreiker, A.: Eingriffe am Auge, Stuttgart, Ferdinand Enke, 1938, p. 124.

disadvantage is its red color and the limit of the size available. Stellwag [3] used vaginal mucous membrane on four occasions only. He abandoned the method, probably because of shrinkage of the graft. By removing the submucosa of this material, Kuhnt [3] obtained less extensive shrinkage. I have used it in a case of severe progressive pemphigus in an elderly woman. The vaginal mucosa healed well, but in three months it resulted in total symblepharon just as in the other eye, on which operation was not performed. Donor and recipient belonged to different blood groups. I tried to obtain epithelization in another case by filling the orbital cavity with vernix caseosa, without any result. Kleitsman and Poska-Teiss [4] restored vagina by this means, getting an epithelized surface after filling the artificially formed cavity several times with vernix caseosa. The authors supposed that embryonal cells of this material adherent to the wall of the cavity would produce a continuous epithelial layer. Grafts from the prepuce or labia minora have been recommended by Clay and Baird.[4a] They used each material with good results. Grafts taken from these areas are thin, contain no hair and little subcutaneous fat and have a pinkish color. Enough tissue was available from the labia minora to restore a socket. These qualities are advantageous indeed, but many would refuse to have the graft taken from such a delicate place.

Not one of these materials is really good from every point of view in replacing the conjunctiva. An ideal material for this purpose should be a thin, smooth, transparent human tissue like the conjunctiva; it should be sterile and obtainable in sufficiently large amount.

Fetal membranes seem to have all these requirements; in addition, they are elastic and carry but few vessels, and the patient does not have to undergo another operation. They have been used by Burger [5] to fit the artificially formed vagina. I obtained such membranes by cesarean section from patients at the Second Women's Clinic, University of Budapest, who had a negative Wassermann reaction. The membranes were kept in tepid Locke solution. One to fifteen hours elapsed between the cesarean section and the implantation. I used this material in 6 cases of symblepharon (8 operations) and in 2 cases in which the socket

3. Cited by Elschnig, A.: Augenärztliche Operationslehre, Berlin, Julius Springer, 1922, vol. 1, p. 410.

4. Kleitsman, R., and Poska-Teiss, L.: Ueber die Anwendung von Vernix caseosa bei der Bildung einer künstlichen Scheide und histologische Untersuchung der letzteren, Zentralbl. f. Gynäk. **59**:755, 1935.

4a. Clay, G. E., and Baird, J. M.: Restoration of the Orbit and Repair of Conjunctival Defects with Graft from the Prepuce and Labia Minora, J. A. M. A. **107**:1122 (Oct. 3) 1936.

5. Burger, K.: Experimental and Clinical Studies on Transplantation of the Fetal Membranes, Orvosi hetil. **82**:800, 1938.

had to be enlarged. Symblepharon was caused in 3 cases by lime burn and in 1 case each by phenol, calcium carbide and pemphigus.

The technic of the operation was the same as proposed by me in 1929.[6] The conjunctiva is undermined from the limbus to the margin of the lid, even if there is but a small portion of fornix left. The undermined conjunctiva is fixed to the lid with two to three mattress sutures to build the palpebral conjunctiva. If there is a complete symblepharon of one lid, I try to line the lower lid with a pedunculated flap of the upper bulbar conjunctiva. While in previous operations I applied mucous membrane of the mouth to cover the defect of the fornix and bulbar conjunctiva produced by the previous procedure, in these 10 operations fetal membranes were used. The graft is fixed to the tendon of one or more rectus muscles, which gives a fixed point on

Result of conjunctival plastic repair with fetal membranes. The patient, a woman aged 28, had had the left eye injured with phenol on May 12, 1938. She was admitted to the State Eye Hospital on July 13. There was no left upper fornix, and the upper edge of the tarsal conjunctiva bent over the limbus. Transplantation of fetal membranes was done on July 21. This picture shows the condition on September 9. The vessels of the bulbar conjunctiva continue in the transplant.

a firm base and avoids shrinkage. The graft covers the fornix, and its edge will unite with that of the conjunctiva lining the lid. The graft is not allowed to make folds but just covers the sclera and fornix smoothly.

When fetal membranes are used the chorion surface is placed on the wound following removal of tense fibers of connective tissue, if any are present. The amnion forms the free surface. In 1 case of total symblepharon of both the upper and the lower lid the fetal membranes were not sutured to the rectus muscles, but a contact glass was covered

6. de Rötth, A.: Bindehautplastik, in Concilium ophthalmologicum, s'Gravenhage (The Hague), Netherlands, 1929.

with the membranes and placed on the eye. The glass was removed on the seventh day when the membranes adhered to the sclera and the lid. In all cases a binocular dressing was applied for three days when the bandage was first changed.

The fetal membranes took in every case, and for three weeks they showed no change. In the next weeks the graft shrunk, and ten weeks after operation it seemed to have disappeared, but there was always more fornix left than before operation. Thus the shrinkage was rather extensive, but the membranes left seem to transform to conjunctiva. However, 1 patient with symblepharon of the upper lid due to phenol had a good result but with a little shrinkage. Three months after operation there was a good upper fornix, as shown in the accompanying illustration. The new vessels of the graft show a course similar to that of the normal conjunctival vessels. One never sees such newly formed vessels in the transplanted mucous membrane. In a case of lime burn the upper fornix had to be restored. The fetal membranes covered the bare upper half of the cornea as well. Ten weeks after operation there was a shallow fornix and the cornea was shiny. In the case of pemphigus the restored lower fornix was shallow two months after the operation. In 3 other cases of symblepharon the late result was poor with nearly total shrinkage of the membrane.

Thus the results were poor or insufficient in but 1 case. The method deserves further study for the following reasons:

1. The embryonal tissue used has a property of being transformed to conjunctiva. The epithelium of the membranes excised two and four months after transplantation showed the same histologic structure as the epithelium of the bulbar conjunctiva.

2. In the successful case the course of the new vessels in the graft was like that of a normal conjunctiva. In no other form of graft may this be observed.

3. The fetal membranes covering the cornea resulted in a shiny transparent tissue. Whether it was the graft itself or whether the graft was replaced by normal corneal epithelium, I do not know.

At present I should confine the indication of using fetal membranes in conjunctival plastic operations to cases of symblepharon when for any reason mucous membrane of the mouth is not obtainable; i. e., when there is disease of the mouth, when the patient refuses operation in the mouth and, further, when the symblepharon includes the cornea as well, for mucous membrane of the mouth gives a poor cosmetic effect when covering the cornea.

SANTA LUCIA, PATRONESS OF THE EYES

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Ophthalmologists who practice in sections where there are many Italians frequently hear reference to Santa Lucia as a patron saint of those who suffer from ocular troubles. Having been presented with a statuette of the saint by a grateful patient, I became interested to learn more of her life and the reasons why she is supposed to make special intercession for those afflicted with disorders of the eyes. It soon became evident that there are both a historical account of Saint Lucy and a legend which sprang up much later, the former entitling her to a place as a virgin martyr and the latter making her a special saint to those with ocular complaints.

Certain facts of the life of Saint Lucy appear to be well authenticated, and she is one of the few women whose names appear in the canon of the mass of Gregory the Great. Born of Christian parents in Syracuse, in southern Italy, at the end of the third century (283), the young girl early evinced profound religious zeal and dedicated her virginity to Christ. Her father having died when Lucy was an infant, she was brought up by her devoted mother, Eutychia. The latter suffered for several years from a flux of blood, which none of the physicians was able to cure. Finally Lucy persuaded her mother to make a pilgrimage to Catania, to the tomb of Saint Agatha, where many miraculous cures were reported to have occurred. While Eutychia was praying at the tomb the hemorrhage ceased, and mother and daughter returned to their home city. Apparently sensing that this was a favorable time to make a request, Lucy persuaded her mother to give her her inheritance, that she might distribute it to the poor. Against her wishes, the beautiful girl had been betrothed to a noble, but pagan, youth of Syracuse, and when the latter learned what she had done with her riches he denounced her to Paschasius, the governor, as a Christian. This occurred during the early part of the fourth century (303), during the reign of Diocletian, when the persecution of the Christians was at its height.

There are numerous accounts of the trial of Lucy before Paschasius, some even recounting verbatim the charges against her, and her replies. In general, it seems believable that she was persecuted as a Christian and that she was condemned to a life of shame. When Paschasius ordered Lucy to be taken away, the crowd of attendants

began to push and jostle her, but she stood immovable. Exasperated, the tormentor commanded that she be carried away by force, but the soldiers were unable to stir her. A yoke of oxen was then brought in, but they also were powerless to move the girl. Certain accounts relate that a fire was built about the victim and that the roaring flames had no more effect than in the case of the three men thrown into the fiery furnace. As a last resort, a sword was drawn and her throat pierced, causing her to bleed to death. As Baring-Gould puts it, "with saints as with witches, when everything else proves ineffectual to hurt them, cold steel breaks the charm."

The account of the life and death of Santa Lucia to this point is generally accepted by church historians, but no mention had been made of any fact that would entitle her to be considered as a protectress of those with afflictions of the eyes. So far as is known there is no early written account of this association, but during the Middle Ages and from then on painters and sculptors have frequently represented Santa Lucia with her eyes on a salver or at times spitted on a sharp instrument. In the Catholic Encyclopedia [1] it is stated "The story of Santa Lucia is to be traced back to the Acta, and these probably belong to the fifth century. Though they cannot be regarded as accurate, there can be no doubt of the veneration that was shown Santa Lucia by the early Church." This source makes no mention of the eyes whatsoever. The first known writer to give an account of the life and passion of Santa Lucia from the Acta was the English bishop, St. Aldhelm of Sherborne, at the end of the seventh century. Since, however, the Acta are admitted to be an untrustworthy source of information, the factual value of the bishop's literary efforts seem slight.

As Mrs. A. B. Jameson stated in "Sacred and Legendary Art," [2] there is no mention in the Acta or in any of the earliest legends of the loss of the eyes. Some of the early painters used the device of placing an emblem of an eye, or eyes, near to her to express her name, Lucia (from the Latin *lux*). From this source seems to have come the invention of the additional incident in her story—"a signal instance of the convergence of the image or metaphor into a fact, which I have so often had occasion to notice."

In the literature there are many references to the story which developed, apparently several hundred years after the historically recorded martyrdom of Santa Lucia, of the manner in which she lost her eyes. The story relates that a youth of her city became enamored

1. St. Lucy, in The Catholic Encyclopedia, New York, The Encyclopedia Press, Inc., 1913, vol. 9, p. 414.

2. Jameson, A. B.: Sacred and Legendary Art, Boston, Houghton Mifflin Company, 1865, vol. 2.

of her and by messages, gifts and all methods known to ardent lovers attempted to win her favor. In his letters and pleadings the young man protested that it was the beauty of her eyes which inflamed him, permitting him no rest by day or night. Fearing that such ardor would be destructive to the youth and that she herself might prove faithless to her vow of chastity, Lucy dwelt on their plight. Recalling the words of Christ, "If thine eye offend thee, pluck it out and cast it from thee," the future saint determined to follow this advice literally and so plucked out her eyes and sent them to her lover on a salver, with the message, "Here thou hast what thou hast so much desired. and for the rest, I beseech thee, leave me now in peace." The story proceeds to a worthy conclusion, for the young man, utterly astonished and torn by remorse and grief, became a convert to Christ and lived ever afterward a life of virtue and chastity.

"But God would not suffer that the blessed Lucia, having given proof of her courage and her piety, should remain blind, for one day, as she knelt in prayer, behold!—her eyes were restored to her more beautiful than before. And if anyone doubts of the great miracle let him consult the writings of that learned and praiseworthy man Fillipo Bergomene, and also of the famous Spaniard Don Juan Moldonato, where they will find it all set down as I have related. And this is the reason that Santa Lucia is invoked against blindness and all diseases of the eyes, and that in her effigy she is represented bearing two eyes on a dish." [2] Here lies the explanation for the curious fact of the martyr sacrificing her eyes by autoenucleation and yet having two other normal and beautful eyes.

Another legend relates that a Roman emperor fell in love with Lucy's beautiful eyes and that when she rejected his suit he caused her eyes to be destroyed. This account is later than the one previously cited and seems to carry no weight.

According to Alban Butler,[3] Santa Lucia was honored at Rome in the sixth century among the most illustrious of the virgin martyrs. Her festival was kept in England until the Reformation as a festival of the second rank, during which no work was done except tillage. In "Weather Folk-Lore" [4] is the statement, "Lucy light, the shortest day and the longest night" (December 13, the day of her martyrdom). Santa Lucia is patroness of Syracuse, of Mantua, of the laboring poor, of tillers of the ground and of sight and the eyes; she is also thought to furnish protection against dysentery and hemorrhage of all kinds.[5]

3. Butler, A.: Lives of the Saints, revised by Herbert Thurston, New York, P. J. Kenedy & Sons, 1926.

4. Swainson, C.: Weather Folk-Lore, London, Trübner & Co., 1885.

5. Dunbar, A. B. C.: Dictionary of Saintly Women, London, George Bell & Sons, 1904, vol. 1, p. 469.

Saint Lucy, worshipped from early times, has been the inspiration of countless artists. In the earliest paintings and sculpture she is shown holding a palm (fig. 1), the symbol of her death for Christianity's sake. In a church in Cajazzo, Italy, there is a twelfth century fresco showing Saint Lucy carrying a lamp, symbolic of illuminating grace. But more often, especially as in later years, she was depicted holding a dish with two eyes on it (fig. 2). More rarely she holds a rod or a dagger which pierces the eyes (fig. 3).

Fig. 1.—Saint Lucy, by Della Robbia. This work, sixteenth century, is to be found in the church of Santa Maria a Ripa, at Empoli, Italy. (Photograph by Alinari.) This and the following illustrations are taken from a small and obviously old book entitled "L'art et les saints—Sainte Lucie," by Georges Goyau, Paris; Henri Laurens, éditeur, 6, Rue de Tournon. On one page, at the bottom, are the words: "Evreux, Imprimerie ch. Hérissey (11-21). No date is given.

There is a manuscript, written in 1220, preserved in the Dome of the Milan Cathedral and signed by Gottofredo of Bussero, stating that Lucy took her name from "light." Cardinal Frederick Borromeo, in the sixteenth century, in his book "On Sacred Paintings" mentioned

a connection between Saint Lucy and the pagan divinity Lucini, who was also recommended for ocular troubles.

Not only have painters and sculptors done homage to Santa Lucia, but great poets and writers have added to the luster of her name. It is stated that Dante wept so much over Beatrice that his eyes were affected and that Saint Lucy, for whom he professed great devotion, cured him. In the "Convivio" he stated that if he had to name two

Fig. 2.—Saint Lucy, by Giovanni Spangna. This painting, sixteenth century, is to be found in the galerie du capitole, at Rome, Italy. (Photograph by Alinari.)

cities he would call one Mary and the other Lucy. In the "Divine Comedy," Dante is represented as distracted by the memory of Beatrice, and from Heaven a "gentle lady," the Virgin Mary in person, saw with pity the soul of the poet so distraught with the death of his love. On Lucy's feast day, December 13, he believed that he heard Mary say to Lucy, "His faith needs your help; I recommend him to you." Whereupon Lucy led Dante and Virgil in the search for Beatrice, and as they traveled over the unfamiliar land they were kept constantly in sight by Lucy. As he fell asleep from fatigue, Lucy appeared to Virgil and

offered to carry Dante on his voyage. In this manner they reached the gateway to Purgatory, when Lucy disappeared. Finally, in Paradise, she reunited Dante and Beatrice. Dante placed Lucy in one of the highest seats in Paradise, in front of Adam, the head of all humanity, and close to the throne of the highest.

Christopher Columbus is related to have been cured of ocular trouble by this patron saint and to have named one of the islands of the

Fig. 3.—Saint Lucy, by Bernardino Luini. This painting, sixteenth century, may be found in the Monasterio Maggiore at Milan, Italy.

Lesser Antilles, discovered on December 13, Santa Lucia, in her honor. Likewise, Allessando Monzoni called one of his principal characters Lucia in his greatest work, "I promessi sposi."

Throughout the Middle Ages, especially in Germany, the night of Saint Lucy, December 13, was thought to be the time when phantoms and demons roamed the earth, spreading ills. Saint Lucy was supposed to be about that night warding off the evil spirits and their malign effects. Even today her feast is observed regularly in Sweden, for on

Fig. 4.—Saint Lucy, by Carlo Dolci. This painting, seventeenth century, hangs in the galerie des offices, at Florence, Italy. (Photograph by Alinari.)

Fig. 5.—Saint Lucy. An engraving from "De claris mulieribres" by J-P. de Bergame.

the dawn of her festival day a girl of each family, dressed in white and wearing a lighted crown, offers coffee and Lusia cakes in honor of the saint, who symbolizes the returning daylight. The most beautiful "Lucia" is selected to lead a triumphal procession in the capital.

The symbolism of a word suggesting definite characteristics is shown in several similar instances. In France Saint Claire is invoked for ocular diseases, because *claire* means clear. In Germany Saint Augustine was supposed to intercede for thoses so afflicted because of his name (suggesting *Auge,* eye).

In the realm of art the earliest paintings of Saint Lucy refer to her trial and martyrdom. It is only later that the symbolism of light and the eyes appears. A painting by Carlo Dolci shows her with rays of light diverging from the wound in her neck (fig. 4), from which she died. Della Robbia represented her with a palm leaf in one hand and a book in the other. The earliest depiction of Saint Lucy known in which there is presented the feature of the loss of the eyes is an engraving by de J.-P. de Bergame, of Ferrara, Italy, in 1497 (fig. 5). A fifteenth century painting by Filippino Lippi, in the Cathedral of Prato, Italy, shows her carrying a lamp in her hand. However, in the sixteenth century the artists almost always pictured her with her eyes on a saucer, and it would therefore seem to be about this period that the legend of the loss and restoration of the eyes became popularly known. A painting by Bernardino Luini in Milan, Italy, shows the eyes held on an awl or similar instrument—a representation not in the best of taste, judged by modern standards.

Among the great painters who have depicted Santa Lucia are: Della Robbia, Antonelle Gaginia, Antonio Riccio, Parmegianino, Bernardino Luini, le Perugin, Lello da Velletri, Carlo Dolci and Giov de Spagna.

Whether or not one believes in the power of saintly martyrs to intercede with the Almighty in the cure of afflictions, there can be no doubt but that much mental comfort and support come to those who feel the need of mediators. And among these saints stands Santa Lucia, who intercedes for those who suffer one of the greatest known afflictions, the loss of sight.

BILATERAL UVEITIS ASSOCIATED WITH DETACHMENT OF THE RETINA (HARADA'S DISEASE)

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The epochal discoveries of Gonin regarding the pathogenesis and surgical treatment of noninflammatory detachment of the retina and the work of various authors in the ensuing years enriched and fundamentally changed the knowledge of this subject. The differentiation between inflammatory and noninflammatory detachment became more distinct. Detachments of the latter type, exhibiting changes in the vitreous and the peripheral part of the retina, occur mainly in persons with myopia and in the senium. The former type, in which the separation of the retina is caused solely by the inflammatory exudation or transudation, is distinguished by the absence of holes or tears. Clinically, in cases of inflammatory detachment one cystlike prominence is usually seen in the lower part of the retina. There is no floating movement present; the detachment, rather, reminds one of a balloon with highly stretched walls. There is a sharp distinction between the two types as to prognosis and treatment, the detachment following or accompanying inflammatory changes in the other membranes of the eye necessitating only symptomatic treatment but no surgical intervention.

Inflammatory changes of a sclerotic nature are known to cause detachment of the retina. Purtscher [1] first observed a case of scleritis complicated by separation of the retina with spontaneous healing. A similar case was described by Kamocki.[2] The condition in each case was unilateral; in Purtscher's case the patient suffered from iritis also. The separation of the retina took place in the area corresponding to the scleral pathologic process and, according to the explanation of Purtscher, was due to the disturbance of the lymph circulation following the infiltration of the sclera. The case of Pichler belongs to the same group. The patient, a man 53 years of age, suffered from recurring scleritis. In the course of one of the serious attacks accompanied by symptoms of periostitis of the adjacent orbital wall, a detachment of the retina established itself, disappearing within seven months. There was no iritis present. The attacks of scleritis in the other eye were not

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1. Purtscher: Centralbl. f. prakt. Augenh. **15**:334, 1891.
2. Kamocki, V.: Centralbl. f. prakt. Augenh. **16**:15, 1892.

combined with retinal detachment. Leber,[3] in his excellent work on retinal disease, mentioned retinal separation in the course of scleritis and episcleritis and tried to explain the mechanism as being similar to that in cases of phlegmon of the orbit, in which the infiltration proceeds along the emissaria to the inner membranes of the eye. The newer literature on the subject contains reports of cases of Horay[4] and Csillag[5] which possibly belong in this group.

Csillag observed a woman 34 years of age with marked scleritis in the upper part of the sclera, ciliary congestion, absence of changes in the anterior segment, a hyperemic disk and detachment of the retina in the upper part of the nasal quadrant without tears or holes. The detachment of the retina progressed, and in the course of the disease the lower and temporal parts were separated too, accompanied by exophthalmos, chemosis of the conjunctiva and serous exudation in the retina, enveloping the vessels. The condition remained unilateral and resulted in complete reattachment of the retina without operative intervention. Doubtlessly the primary disease, the circumscribed scleritis, was instrumental in producing the detachment in the corresponding area of the retina. With the progress of the inflammatory changes, the detachment occupied the lower and temporal parts of the retina as well. The explanation given was that the acute inflammatory reaction caused edema of the choroid, the stasis of tissue fluid sank downward due to the loose consistency of the choroidal structure and the increased volume resulted in detachment of the retina. Further proof of the mechanism of edema is given by the fact that after absorption of the fluid and disappearance of the edema, the fundus did not reveal any pathologic changes referable to previous choroidal inflammation.

The chapter dealing with retinal detachments in the course of deep scleritis was further enriched by a contribution of Karasek,[6] in whose case posterior scleritis led to neuritis, then retrobulbar neuritis, paresis of the abducens nerve and a rather flat retinal detachment of the macular region (about +3.0 diopters), which is explained by exudation between the sclera and the choroid with an ensuing inflammatory involvement of the choroid and retina due to the infectious toxic substances.

Meisner[7] contributed 4 cases of his own to this review, emphasizing the rarity of cases and the favorable prognosis. Among 150 cases of detachment, he observed only 4 of the inflammatory type in which

3. Leber, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1915.
4. Horay, G.: Klin. Monatsbl. f. Augenh. **95**:656, 1935.
5. Csillag, F.: Klin. Monatsbl. f. Augenh. **98**:206, 1937.
6. Karasek, O.: Klin. Monatsbl. f. Augenh. **95**:645, 1935.
7. Meisner, W.: Schweiz. med. Wchnschr. **67**:788, 1937.

surgical intervention was not indicated and the symptomatic treatment resulted in complete healing. In his first case, that of an emmetropic woman 40 years of age, there was a detachment of four weeks' duration occupying the lower temporal part of the retina. No holes or folds were present, and there were a few small old choroidal patches at the margin of the detachment. The cutaneous test for tuberculosis was positive. After reattachment, visible changes of the choroid in the macular area took place. The case is classified as one of tuberculous choroiditis with retinal detachment.

The patient in the second case gave a history of painful scleritis of many months' duration before a large detachment developed in the lower part of the retina. There were no ruptures and no inflammation in the anterior part of the eye. Histologic examination of the enucleated eye revealed the presence of a tumor of the sclera of fibromatous appearance, small foci of calification, infiltration suggesting a tuberculous origin and similar changes in the posterior part of the sclera and choroid.

The patient in the third case, a woman 30 years of age, showed marked ciliary congestion of the right eye, increased corpuscular elements in the aqueous, numerous delicate deposits on the anterior capsule, fibrinous masses at the dilated pupillary margin, opacities in the vitreous, a hazy and swollen disk and detachment in the lower part of the retina without the formation of folds. The left eye, simultaneously, was affected by similar, but less marked, inflammatory changes in the anterior segment without retinal separation. As the disease advanced, fibrinous nodules appeared in the left iris and a detachment of the retina in the lower temporal part resulted. After healing, depigmentation and numerous smaller and larger pigment patches at the site of the previous detachment were evident. Serologic and tuberculin tests were negative.

The patient in the fourth case, a man 54 years of age, suffered from rheumatism and recurring iridocyclitis in both eyes. The right eye showed ciliary congestion, pigmentation of the anterior capsule, a normal iris, a clear vitreous and an extensive detachment of the entire lower part of the retina without tears. The other eye was normal, only remnants of previous iritis being discernible.

All 4 patients had in common retinal detachment as a sequel to inflammatory changes in other membranes of the eyes. With the exception of the third patient, all showed unilateral involvement. The first presented tuberculous choroiditis; the second, severe tuberculous involvement of the sclera and choroid, and the third and fourth, inflammatory changes of the uvea, the iris and the ciliary body. The detachment of the retina developed in the course of acute inflammatory

reaction and healed under symptomatic treatment without the necessity of surgical aid. The resorption of the exudation resulted in complete reattachment.

The unilateral, purely inflammatory retinal detachment forms the bridge to the other type of detachments in which the separation is simultaneously bilateral and is the main feature of the symptom complex. The detachment is so much the important aspect of the pathologic process that the other possible inflammatory symptoms are apparently of lesser significance. Harada [8] in 1926 first described bilateral detachment in the course of low grade uveitis as an independent disease entity. The first publication came from Japanese sources only (12 cases). It seems that Salus [9] was the first European to publish an observation belonging to this group. Harada's disease presents an acute, diffuse, exudative choroiditis or uveitis bordering on diffuse uveitis accompanied by deafness, poliosis and loss of hair and in turn on sympathetic uveitis. However, the typical onset, the extensive retinal detachment and the relatively favorable prognosis of the spontaneous reattachment are features warranting the consideration of the separation as an entity. Harada's disease is invariably bilateral; the onset is a rather acute one accompanied by headaches and occasional vomiting or nausea and characterized by either lack of, or low grade, inflammatory symptoms in the anterior segment of the uvea. In the early stage, opacities of the vitreous, edema of the disk, suggestive of papillitis, and yellowish discoloration of the retina are present; these are followed after a few weeks' duration by extensive detachment. The detachment has a definite tendency to disappear gradually within a few weeks, and the healing results in unexpected good visual acuity, in view of the previous extensive and severe pathologic changes. The eyeground picture is that of depigmentation, the redness becoming lighter with the presence of patches of pigment in the retina due to proliferation of pigment elements.

A patient of Salus, a woman 38 years of age, had headaches at the onset; the eyes were white, but numerous delicate grayish white deposits were present, especially in the right eye. The irides exhibited small nodules or vesicles similar to those seen in cases of tuberculosis of the iris. Delicate opacities of the vitreous, markedly swollen disks and enormously dilated and tortuous retinal veins were present. Yellowish gray cloudiness was observed in the retina. In the course of the disease posterior synechiae appeared, the nodules of the irides disappeared and the retinal infiltration became more extensive. In the seventh week of the disease, simultaneous detachment of the retina occurred in each eye, progressing from the periphery toward, and finally reaching, the

8. Harada, cited by Salus.[9]

9. Salus, R.: Klin. Monatsbl. f. Augenh. **89**:84, 1932.

disk. The detachment disappeared about the same time in each eye simultaneously with the other inflammatory symptoms (scleral puncture was performed on the left eye). After the disappearance of the latter, numerous posterior synechiae with a delicate occlusion membrane were present. The fundi appeared albinotic due to the depigmentation, with visible choroidal vessels not exhibiting any vascular pathologic process, but a few proliferative patches of pigment were noted. In the periphery there were a few small white patches suggestive of sympathetic choroiditis and at the posterior pole wide stripes of organized exudate with delicate pigmentation. The case belongs, according to Salus, to the group of cases of Harada's disease, notwithstanding the few apparent discrepancies, such as pathologic involvement of the anterior segments marked by the presence of deposits on the posterior surface of each cornea and nodules in the irides, which he did not consider tuberculous, the intracutaneous tuberculin test showing only a slight reaction.

Salus' case appears more remarkable because of the interesting fact that a sister of the patient two months later suffered from similar iridocyclitis with formation of nodules in the irides. The disease appeared simultaneously in each eye and involved almost exclusively the anterior parts only, the choroid remaining free of changes with the exception of a few small peripheral patches.

Horay's patient, a man 46 years of age, had in the left eye opacities in the vitreous, detachment of the retina in the lower part without tears and chorioretinitic patches in the periphery; the detachment flattened out ten days after scleral coagulation. A short time later a scleritic nodule developed in the normal right eye; later there developed chemosis of the conjunctiva, periosteal irritation at the temporal part of the orbit and detachment of the retina corresponding in area to that of the scleral infiltration. Later, there were visible exophthalmos and total detachment of the retina. The detachment disappeared without operative intervention. Reexamination after three months showed a picture of hazy margins of the disk, dilated retinal veins and a yellowish red retina with patches of pigment; in the region of the ora serrata, a flat, ring-shaped, almost imperceptible detachment remained.

The extreme rarity of similar observations and the somewhat meager reports in the literature have prompted the publication of 2 cases which, doubtlessly, belong in the group of cases under discussion and demonstrate that even within the same group the prognosis and ultimate outcome of the given case may show extensive variations.

REPORT OF CASES

CASE 1.—A man 22 years of age, of Italian parentage, was first seen on Oct. 30, 1937. He complained of progressive loss of vision for the preceding six weeks. He had had "grip" seven weeks prior to the onset of blurred vision. About three

days after his recovery from "grip" he noticed loss of vision on the nasal side of his left eye. The vision became progressively worse in both eyes, so that at the end of four weeks he retained only perception of light and was unable to verify objects.

He could not recall having had any diseases of childhood. There was no history of cardiac, respiratory or metabolic disease or of syphilis or gonorrhea. The family history did not reveal any ocular disease. There was no history of tuberculosis. He had fractured his right arm many years ago.

Physical examination showed a well developed white man. The tongue was moist, and the pharynx was not injected. There were a few small movable nodes in the neck and no enlargement of the thyroid. The heart, lungs and abdomen were normal. The reflexes were physiologic.

The right palpebral fissure was smaller than the left. Each eye showed conjunctival and slight ciliary congestion. The endothelial layer showed marked edema. The lower part of the cornea had a tissue paper-like appearance with a few small round grayish deposits. The irides were somewhat swollen, and there were no newly formed capillaries. The pupils dilated, but not to the maximum (atropine); the diameter of each pupil was about 5 mm. On the anterior capsules there was delicate pigment dust, and on the area corresponding to the undilated pupillary margin, larger patches of pigment. The vitreous in each eye was very hazy, the haziness being of a diffuse character. There were no large floaters, only a dustlike variety. There was an enormously extensive retinal detachment with a maximum prominence of + 16.0 diopters in the right eye, involving the nasal, lower and temporal parts of the retina, and in the left eye involving the lower and nasal parts of the retina.

Roentgen examinations were made (Dr. J. Furst). The accessory nasal sinuses showed insufficient evidence of gross pathologic change to be of diagnostic value. There was, perhaps, a slight degree of congestion of the left ethmoid sinus. Exposures of the thorax showed no evidence of active parenchymal infiltration or pleural involvement.

The urine was clear and acid and had a specific gravity of 1.202; there was no albumin or dextrose. The blood count showed: 12,200 leukocytes, 73 per cent polymorphonuclears, 12 per cent lymphocytes, 8 per cent endothelial leukocytes, 4 per cent eosinophils, 1 per cent basophils, 68 per cent hemoglobin, and 3,850,000 erythrocytes. The urea nitrogen content was 132 mg.; the uric acid, 32 mg.; the creatinine, 1.3 mg., and the sugar, 88 mg. per hundred cubic centimeters. The Wassermann and Kline reactions were negative. The sedimentation rate of the blood was 50 mm. the first hour and 80 mm. the second hour. Cultures from the right and left nostril showed Staphylococcus albus; from the throat, Streptococcus haemolyticus and Micrococcus catarrhalis. A filtrate (0.2 cc.) of Str. haemolyticus given intradermally caused no reaction. The Mantoux test was negative.

Within the next ten days, the ciliary congestion cleared up completely. Both eyes became extremely hypotonic. The irides in the peripheral parts were almost touching the posterior corneal surfaces due to multiple circumscribed bulging areas; the pupillary part of each iris was drawn backward toward the vitreous. The backward pull was not funnel shaped but almost at an angle of 90 degrees, especially in the nasal part of the right eye.

Within the ensuing seven days numerous large deposits appeared all over the posterior corneal surface of the right eye. Only a few small deposits were seen in the lower part of the left eye. The pupillary margin in each eye was circularly attached to the lens with a few delicate capillaries growing from the iris into the thin seclusion membrane, covering the entire pupillary area. The anterior chambers

were extremely deep, the lens-iris diaphragms being drawn back toward the vitreous. A few grayish nodules were seen in the irides. The fundi were invisible. Hypotonia of extreme degree persisted.

Within the next four weeks vision in each eye improved to counting of fingers at 1 meter. The right disk was hazy and the margins veiled. In the nasal part there was delicate pigmentation and in the lower part, numerous round white pinhead-sized patches arranged in grapelike clusters. There were a few large opacities in the vitreous of the left eye and delicate pigmentation in the retina. The previously extensive detachment of the retina completely disappeared.

Two months later the right eye showed slight ciliary congestion but a markedly increased number of deposits; the entire posterior surface was covered, carpet-like, with large round yellowish deposits. The left eye did not show any ciliary congestion but more deposits. The vitreous in each eye was diffusely hazy, but the fundi were clearly visible. The fundi were yellowish and had less vivid redness than usual. Complete reattachment of the retinas occurred. The disks were somewhat veiled, and the veins were dilated and engorged. Over the retinas there were small irregularly outlined patches of brownish pigment, occasionally covering the retinal vessels; their relation to the retinal vessels revealed the different localization in the various layers of the retinas. In some parts of the retinas there were pinpoint to pinhead-sized white patches with delicate, rather black pigmentation similar to the changes occurring in cases of healed sympathetic choroiditis. The vision in the right eye was 20/200 and in the left eye 20/50; the patient did not accept glasses. The tension (Schiötz) in the right eye was 10 mm. of mercury and in the left eye 12 mm.

The patient was seen again two months later. The inflammatory symptoms in the anterior segments had not increased, but the fundi were invisible because of rapidly developing complicated cataracts. Hypotonia persisted; the vascularization of the seclusion membrane was more marked than before.

General treatment consisted of injections of foreign protein intramuscularly, followed by mercury inunctions and tuberculin.

Case 2.—A girl 18 years of age, of Italian extraction, noticed a sudden diminution of vision three weeks before examinaation. There was pain in each eye at the same time, and the eyeballs turned very red. General examination gave negative results.

The urine was clear and showed an acid reaction, a specific gravity of 1.023, 2 mg. of albumin, no dextrose or casts and 15 leukocytes (high power field) and 2 epithelial cells. The blood count revealed: 8,700 leukocytes, 70 per cent polymorphonuclears, 19 per cent lymphocytes, 5 per cent endothelial leukocytes, 1 per cent basophils, 3 per cent myelocytes (stab forms), 81 per cent hemoglobin and 4,200,000 erythrocytes. The urea nitrogen content was 20 mg. and the blood sugar 100 mg. per hundred cubic centimeters. The Wassermann and Kline reactions were negative. The sputum was negative.

Roentgen examinations were made (Dr. J. Furst). The accessory nasal sinuses showed fairly good aeration and ventilation. An exposure of the thorax showed insufficient evidence of gross pathologic changes to be of diagnostic value.

The reaction to the Mantoux test (with a 1:1,000 dilution of tuberculin) was 3 plus. (After twenty-four hours there was redness and induration 0.5 cm. in diameter.)

Vision in the right eye was 15/200 and in the left 15/100; there was no improvement with glasses. Marked conjunctival and ciliary congestion was present in each eye. The slit lamp showed the posterior surface of each cornea to be tissue

Fig. 1 (case 2).—Right eyeground. This illustration and also figure 2 were made about three months after reattachment of the retina took place. It is interesting to note the yellowish hue due to depigmentation. The small pigment patches are of a brownish color and aggregate in the macular region. Since the drawing of the pictures, the patches have increased somewhat in number but not in size. On both eyegrounds a stripe-shaped pigmented line, somewhat elevated, is clearly visible.

Fig. 2.—Left eyeground of the same patient.

UNIV. OF MICH.

paper-like and covered with delicate fibrinous stripes. The anterior chamber of the right eye contained fibrinous exudation. There were numerous hyperemic posterior synechiae in the irides, especially in the upper part of the right iris. No deposits were noted. A delicate fibrinous membrane was present in both pupillary areas. There was an extensive retinal detachment in the lower part of each retina; the maximum prominence in the right retina was + 8.0 diopters and in the left, + 12.0 diopters. The detachment was more extensive in the left eye than in the right. The vitreous in each eye was diffusely hazy, and the retinal veins were markedly dilated and engorged.

Nine days after the initial examination there were a few deposits on the right eye and more deposits in the lower part of the cornea of the left eye. The deposits were white and star shaped. The pupils dilated to the maximum. There were almost circular posterior synechiae and increased circulation and pigmentation of the anterior capsules. The anterior chambers were deep and the irides markedly retracted. The tension (Schiötz) was 12 mm. of mercury in each eye.

Fifteen days later the deposits were markedly increased in number and size. In the right eye, temporal from the disk, there was one large stripe-shaped area of retinal hemorrhage. Complete reattachment of the detached part of the retina had occurred in each eye.

Three months later both eyes were white. There were no symptoms of irritation or synechiae, and the vitreous was clear. Vision in the right eye was 15/30; with a + 0.5 sph. $\bigcirc$ + 0.5 cyl., axis 180 it was 15/15. Vision in the left eye was 15/30; with a + 0.25 sph. $\bigcirc$ + 0.5 cyl., axis 165 it was 15/20. The tension (Schiötz) was 22 mm. of mercury in each eye.

Figures 1 and 2 show the condition observed in the eyegrounds. The fundi were definitely pale, somewhat yellowish in comparison with the usual red color. On the fundi, especially in the macular region of each eye, numerous small brownish patches of pigment were visible. They were marked by the brownish hue in contradistinction to the black color of pigment usually present in the pigment frame or in overpigmentation in the atrophic patches of old choroiditic lesions. The distribution was characterized by the fact that most of the patches were located in the macular region and there were only a few patches in the periphery. The patches were small, round or oval, without sharp outlines. In each eyeground one pigmented stripe was visible; in the course of the stripe, the retinal vessels were both underbridged and overbridged, denoting that the stripe was located in different layers of the retina.

COMMENT

It is important to note that in both cases there were a sudden onset, bilateral involvement, low grade uveitis and detachment of the retinas which healed without surgical intervention. The detachments in each case were rather extensive, and no holes or tears were present. The clinical course, however, differed widely in the two cases. In the first, the patient had "grip" before the onset of the ocular symptoms. The retraction of the iris-lens diaphragms and the long-lasting and extreme hypotonia warranted an unfavorable prognosis from the start. After reattachment of the retinas, the acute inflammatory symptoms continued and gained in severity. The deposits increased in number and size, and the seclusion membrane became thicker and vascularized, finally leading to the formation of complicated cataracts. The uveitis presented

itself in a most severe form, a type which is usually encountered in the group of sympathetic uveitis or inflammations closely related to it by their severity.

In the second case the inflammatory symptoms of the uvea were distinctly milder. In the early stages there were no deposits discernible, not even under slit lamp examination. The picture was within the framework of fibrinous iritis and was accompanied by deposits only later. After reattachment of the retinas, the entire inflammation subsided, the hypotonia disappeared and normal vision in one eye and almost normal vision in the other resulted. The retinal changes were similar in the two cases. In the second case the changes consisted of pigment proliferation within the retinas; in the first case, in addition to the proliferation, delicate, pointlike, white atrophic patches were noted in the choroid of each eye.

The sudden bilateral appearance in young persons of uveitis complicated with extensive retinal detachment markedly circumscribed the pathologic process in these cases. The careful clinical and laboratory analyses did not reveal any data of value concerning the cause of the disorder. In the first case the clinical appearance of the uveitis was of a much more severe type and of much longer duration than in the second.

Doubtlessly uveitis complicated with detachment with sudden onset and simultaneous appearance in each eye represents an entity in itself. Next to the bilateral appearance, the usual location in the lower part of the retina and the absence of holes or tears are significant. The latter condition is a usual finding in the inflammatory type of separation of the retina. But other types of inflammatory detachment may show tears occasionally, as I had occasion to observe in a case of unilateral tuberculous iridocyclitis in a young person, in which case, about three months after the acute iridocyclitis, a large detachment with a semicircular tear presented itself, and diathermic coagulation of the tear produced a perfect and lasting result. In bilateral uveitis associated with detachment there seems to be no interruption in the continuity of the retina, and, accordingly, the healing and reattachment are achieved without surgical intervention. The usual unilateral inflammatory type of detachment, like that caused by scleritis or tuberculous choroiditis, is present and makes its appearance almost simultaneously in each eye.

The Japanese author Harada was the first, in 1926, to classify this group of conditions as a separate disease entity. Important are the bilateral appearance, the acute onset with headaches, occasional nausea and vomiting and the low grade inflammatory changes in the anterior segment of the uvea (or possibly, complete absence). Ophthalmicroscopic examination reveals opacities of the vitreous and a diffuse

yellowish color of the retina with papillitis-like changes of the disk. After a few weeks extensive and bilateral detachment is noticeable and is followed by spontaneous healing and surprisingly good function, considering the pathologic involvement previously present. The end result presents depigmentation with pigment proliferation.

Takahashi [10] used inoculation of the vitreous fluid in rabbits. The fluid did not contain pathologic micro-organisms. With intracisternal inoculation, in 2 of 5 cases he produced a descending optic neuritis and uveitis. The inoculation of the cerebrospinal fluid of the patient into the vitreous of rabbits produced inflammation in 1 of 4 cases. On inoculation of the cistern with brain tissue of infected animals, optic neuritis and iridocyclitis were produced; the direct inoculation into the eye produced inflammation of each eye by the route of the optic nerve. According to the results of inoculation, the disease should be classified as infectious, belonging in the category of sympathetic and herpetic lesions. Tagami's [11] inoculation of the vitreous of rabbits with subretinal fluid similarly resulted in uveitis, which histologically resembled sympathetic uveitis with the exception of giant cells. The infiltration in the anterior parts of the uvea was more marked; the infiltration of the choroid consisted of large round mononuclear elements. Furthermore, detachment of the choroid and retina through serofibrinous exudate and degeneration of the pigment epithelium with almost negligible changes within the retina took place. Accordingly, the disease, which seems to be more prevalent in Japan, should be classified with the virus group of diseases.

Contrary to these views are the results of Oka-Mura [12] who reported about 2 cases in his first, and 16 cases, in his second publication. The slit lamp revealed in the first case the presence of miliary nodules in the iris in large numbers, and a few nodules were discernible in the second case. Secondary glaucoma necessitated the performance of iridectomy in the first case, and histologic examination of the iris showed giant cells of Langhans type and therefore a similarity with sympathetic or tuberculous structure. In the second case the histologic findings should be analogous with those of beginning sympathetic lesions. Roentgenograms of the lungs, the sedimentation test and the general examination revealed tuberculosis. In the second publication Okamura considered all the cases of tuberculous origin. The nodules in the iris, the disseminated atrophy of the choroid, the perivasculitis of the retina and the bleeding into the vitreous are manifestations of that disease.

10. Takahashi, M.: Acta Soc. ophth. jap. **34**:33, 1930.
11. Tagami, K.: Acta Soc. ophth. jap. **35**:114, 1931.
12. Oka-Mura: Acta Soc. ophth. jap. **41**:679, 1937; **42**:196, 1938.

The discrepancy in the mechanism between virus disease and tuberculous origin is widespread and cannot be answered at present dogmatically. Further observation of similar cases will help greatly to solve the problem.

The clinical analysis shows that in smaller details there is a certain variation in the clinical picture presented. In the case of Salus the deposits and the nodules of the iris indicate a more active participation of the anterior segment of the uvea, a circumstance which could be interpreted in the light of a tuberculous origin. Salus was not inclined to consider it as such but emphasized the possible infectious character, citing the circumstance that the sister living with the patient suffered from a similar severe type of uveitis two months later. The case of Horay is more complex and therefore more difficult to classify. The absence of inflammatory symptoms in the anterior part, the bilateral (but not simultaneous) detachment with spontaneous healing and the resulting changes of the fundi are arguments in favor of classifying the condition as Harada's disease. The presence of marked scleritis and the development of a detachment, on the other hand, would place it in the group of cases of scleritis associated with retinal separation. In this group even repeated detachments are possible phenomena, as the recent publication of Zenker [13] shows.

In the chapter on inflammatory detachments, the cases of bilateral simultaneous detachment accompanying uveitis doubtlessly represent a distinct and separate entity. The clinical picture differs from that of idiopathic detachment by the absence of holes and tears and is closely allied with that of unilateral detachment associated with scleral inflammation by the tendency to spontaneous healing. The depigmentation of the fundus and the proliferative pigment changes after healing has taken place are further characteristics of the entity. The primary cause leading to detachment is the uveitis, which seems to differ in intensity and expansion, as demonstrated by the severity in my first case and the relatively mild course of the second one. Similarly, in the cases published in the literature the uveitis is of various degrees; in some cases only the posterior segment of the uvea is involved, and in other cases the uveitis extends into the anterior segment. The inflammation of the anterior segment results in symptoms presenting various forms and degrees of inflammatory reaction. The underlying pathologic explanation is not held unanimously and varies all the way from that of a virus disease to a possible tuberculous origin.

31 Lincoln Park.

13. Zenker, C.: Klin. Monatsbl. f. Augenh. **102**:429, 1939.

RELATION OF DYNAMIC TO STATIC REFRACTION IN PRESBYOPIC PATIENTS FORTY THROUGH FIFTY YEARS OF AGE

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Although it is commonly recognized among ophthalmologists that the use of a cycloplegic drug is a necessity in the refraction of all persons who have not reached the age of presbyopia, yet it cannot be said that any truly general rule exists with respect to the use of cycloplegics in the refraction of presbyopic patients.

Many refractionists regard it as unnecessary to inconvenience their patients by the administration of a cycloplegic when the presence of presbyopia makes manifest refraction feasible. Others, of no less repute, do not believe that refraction can be done with scientific precision for even the most presbyopic person without cycloplegia.

The purpose of this study was to make a careful measurement of the refraction under dynamic and under static conditions and to compare the results in order definitely to demonstrate whether dynamic methods can be expected to uncover the full static correction for the average patient in the early stages of presbyopia.

The material for this research consisted of 100 persons with early presbyopia from 40 through 50 years of age who applied to the ophthalmic clinic of the Indianapolis City Hospital for refraction. When each patient was first seen, the refraction was determined by the manifest method by means of fogging. Then eucatropine hydrochloride in a 3 per cent solution was instilled into each eye every five minutes until a total of 5 drops (or more if required to obtain mydriasis) was administered. Thirty minutes after the instillation of the last drop refraction was done by retinoscopic and by trial case examination (retinoscopic examination alone being done for 25 of the 100 patients). At least seven days later each patient returned to the clinic after having instilled into his eyes at intervals of ten minutes a total of 10 drops of a 4 per cent solution of homatropine hydrobromide. Refraction was then done by retinoscopic and by trial case examination. No less

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then seven days after refraction with homatropine a postcycloplegic examination was made, at which time the addition required for near vision was determined. After refraction with homatropine, the far point of 91 of the patients was measured after a + 3 diopter sphere had been added to the infinity correction. The near and far points of 36 persons were both thus measured after manifest, eucatropine and homatropine refractions.

EXPERIMENTAL DATA

Refraction with homatropine is accepted as being the most accurate of the three methods. Therefore, the results of refraction by the manifest method or by retinoscopic and by trial case examination with the eye under the influence of eucatropine are directly compared with the results of refraction with the eye under the influence of homatropine.

The relation of refraction by the manifest method and with eucatropine to refraction with homatropine is of interest in the extent to which the algebraic sum of the sphere and cylinder in each case agree and in the degree of accord between the amount of cylinder and the position of the cylinder axis as found by the several methods. Of the two items, the latter is much the more important, for, after refraction with homatropine it is usually necessary to deduct a portion of the sphere in order to allow the accommodation a small amount of latitude in infinite vision. Therefore, the spherical element in the prescription is, in the age group of this study, of no great significance so long as it reasonably approximates the true sphere as uncovered by refraction with the use of a cycloplegic. Of much more significance is the algebraic sum of the sphere and cylinder, but this statistic, too, is influenced by possible deductions from the prescription. Hence, the chief source of information in the comparison of the three methods of refraction must be the statistics which concern the cylinder. Analyses of the algebraic sum of the sphere and cylinder and of the cylinder only will be presented, but all conclusions will be based entirely on the latter. The results with the manifest method are shown in the following outline:

Algebraic Sum of Sphere and Cylinder

Increase over that found with refraction with homatropine in 61 of 200 eyes, or 30.5 per cent

Average increase, 0.381 diopter

Decrease below that found with refraction with homatropine in 110 of 200 eyes, or 55 per cent

Average decrease, 0.614 diopter

No change from that found with refraction with homatropine in 29 of 200 eyes, or 14.5 per cent

Amount of Cylinder and Deviation of Axis

Increase in the amount of cylinder over that found with refraction with homatropine in 36 of 200 eyes, or 18 per cent

Average increase, 0.317 diopter

Decrease in the amount of cylinder below that found with refraction with homatropine in 46 of 200 eyes, or 23 per cent

Average decrease, 0.692 diopter

Deviation of the cylinder axis in 87 of 200 eyes, or 43.5 per cent

Average deviation, 28.44 degrees

No change in the amount of cylinder or in the axis in 47 of 200 eyes, or 23.5 per cent

Cylinder uncovered by refraction with homatropine which was not detected by manifest refraction in 25 of 200 eyes, or 12.5 per cent

Average amount of latent cylinder, 0.415 diopter

Cylinder uncovered by manifest refraction which could not be detected by refraction with homatropine in 24 of 200 eyes, or 12 per cent

Average amount of false cylinder, 0.468 diopter

Deviation of the cylinder axis the only change in 22 of 200 eyes, or 11 per cent

The most striking fact which these data bring out is the high incidence of increase in the refractive error as determined by the manifest method. The algebraic sum showed such an increase in 30.5 per cent of all eyes examined, while the amount of cylinder was increased in 18 per cent. The average increase in the amount of cylinder, 0.317 diopter, is significant. This phenomenon may be accounted for by the fact that refraction by the manifest method is performed with the vision fogged, the correction accepted being that which permits the accurate discrimination of the letters in the 6/6 line with the greatest amount of plus lens. Under such circumstances it is conceivable that certain persons with good visual acuity, even with an increase in the amount of cylinder of 0.317 diopter, will readily be able to recognize the test letter even though the retinal image is not perfectly sharp.

A decrease in the amount of the cylinder occurred in 23 per cent of the eyes examined. As a decrease is rather the expected variation, it is surprising that its occurrence exceeded that of an increase by only 5 per cent of the eyes refracted. However, as might be expected, the average amount of decrease exceeded that of increase by better than 2 to 1. Certainly a cylinder which is estimated 0.692 diopter short of the true correction cannot be considered as representing excellence in refraction.

Forty-three and one-half per cent of all eyes examined showed the cylinder axes to have deviated an average of 28.44 degrees from their location at cycloplegic refraction. This constitutes a high percentage

of failure, for no refractionist cares to acknowledge an inability to locate the cylinder axis within such wide limits. Five degrees might be tolerated, but hardly 28!

Of the total number of eyes, 12.5 per cent showed a failure of the manifest method to uncover cylindric error of an average amount of 0.415 diopter, while 12 per cent showed the presence of cylindric error averaging 0.468 diopter which could not subsequently be confirmed by cycloplegic examination. Thus, the correction of the cylindric error was a complete failure in 24.5 per cent of all eyes refracted by the manifest method.

Not even in one fourth of the eyes refracted was the manifest method successful in revealing the true amount of cylinder and its axis. Such accuracy was found in only 23.5 per cent of the 200 eyes.

The results of refraction with eucatropine are shown in the following outline:

Algebraic Sum of Sphere and Cylinder

Increase over that found with refraction with homatropine in 84 of 200 eyes, or 42 per cent

Average increase, 0.331 diopter

Decrease below that found with refraction with homatropine in 82 of 200 eyes, or 41 per cent

Average decrease, 0.462 diopter

No change from that found with refraction with homatropine in 34 of 200 eyes, or 17 per cent

Amount of Cylinder and Deviation of Axis

Increase in the amount of cylinder over that found with refraction with homatropine in 25 of 200 eyes, or 12.5 per cent

Average increase, 0.338 diopter

Decrease in the amount of cylinder below that found with refraction with homatropine in 44 of 200 eyes, or 22 per cent

Average decrease, 0.477 diopter

Deviation of the cylinder axis in 64 of 200 eyes, or 32 per cent

Average deviation, 38.81 degrees

No change in the amount of cylinder or in the axis in 22 of 200 eyes, or 11 per cent

Cylinder uncovered by refraction with homatropine which was not detected by refraction with eucatropine in 33 of 200 eyes, or 16.5 per cent

Average amount of latent cylinder, 0.404 diopter

Cylinder uncovered by refraction with eucatropine which could not be confirmed by refraction with homatropine in 17 of 200 eyes, or 8.5 per cent.

Average amount of false cylinder, 0.382 diopter

Deviation of the cylinder axis the only change in 22 of 200 eyes, or 11 per cent

As is the case with the manifest method, there was a high incidence of increase in the refractive error when eucatropine was used. The algebraic sum showed an increase in refractive error in 42 per cent of the eyes examined. This is 11.5 per cent more than with the manifest method. On the other hand, the amount of the cylinder showed an increase in only 12.5 per cent of the eyes as compared to 18 per cent with the manifest method.

Decrease in the amount of the cylinder occurred in 22 per cent of the 200 eyes, which is 1 per cent less than the figure obtained by the manifest method. This, plus the fact that the average amount of decrease was distinctly less (0.215 diopter) than that found under refraction by the manifest method, suggests that the refraction with eucatropine is regularly uncovering a greater proportion of the error than the manifest method. Nevertheless, 0.477 diopter represents too great a deficiency in the amount of the cylinder to be consistent with true refractive accuracy.

Deviation of the cylinder axes occurred in 32 per cent of 200 eyes, representing a reduction of 11.5 per cent from that found with the manifest method. The average deviation is, however, 10.37 degrees greater with refraction with eucatropine. Although the latter method does represent some improvement on this point, the incidence of deviation is far too high and the average deviation of 38.81 degrees is intolerable to most patients.

Refraction with eucatropine failed to uncover cylindric error of an average amount of 0.404 diopter for 16.5 per cent of the 200 eyes examined, while for 8.5 per cent the presence of cylindric error averaging 0.382 diopter was uncovered which could not subsequently be confirmed by cycloplegic examination. This represents complete failure in correcting the cylindric error in a total of 25 per cent of all refractions with eucatropine. The average amount of the cylindric error not uncovered shows little variation from that reported with the manifest method, while the amount of false cylindric error uncovered shows a decrease in average amount of 0.086 diopter. As a whole, the record for refraction with eucatropine is no better than that for the manifest method of refraction in terms of complete failure in detecting cylindric error.

It would appear that, considering all of the data, the refraction with eucatropine represents some improvement over the manifest method in that it more closely approximates the refraction with homatropine cycloplegia. The explanation of this greater accuracy is to be found either in the fact that the use of eucatropine permits objective analysis by retinoscopic examination or in a cycloplegic property of the drug

or in a combination of both factors. No accurate information can be offered as to the true situation, but it is possible definitely to demonstrate that, contrary to the accepted pharmacology, eucatropine is a weak cycloplegic. Thirty-six patients, representing 72 eyes, had their puncta proxima [1] measured with a plus 3 diopter lens added to the distance correction after each refraction was performed. Comparison of the puncta proxima after refraction by the manifest method with that found after refraction with eucatropine gives evidence of the development of cycloplegia after the use of eucatropine in 46 of the 72 eyes, or in 64 per cent.

1. Increase in the total plus lens with recession of the puncta proxima ..... 34 eyes, or 47.2 per cent
2. Increase in the total plus lens with no change in the position of the puncta proxima..... 10 eyes, or 14 per cent
3. No change in the total plus lens with recession of the puncta proxima ..... 2 eyes, or 2.8 per cent

These data can leave no doubt that some degree of cycloplegia is induced by the use of eucatropine and plays a definite part in enhancing the efficacy of this type of refraction. The experiment, however, has not been controlled in such a fashion as to permit any deduction concerning the relative importance of cycloplegia as compared to objective examination with the retinoscope. It must, therefore, suffice that both factors are definitely present, their exact relation remaining, for the present, unknown.

Of considerable interest is the distribution of depth of presbyopia among the cases analyzed. The additions prescribed at the time of the postcycloplegic examination afford an accurate indication of the state of presbyopia in each case:

1. No addition required..... 13 per cent of cases
2. An addition no greater than 1 diopter required..... 26 per cent of cases
3. An addition of more than 1 diopter but no greater than 2 diopters required ..... 45 per cent of cases
4. An addition greater than 2 diopters required..... 16 per cent of cases

It now becomes evident that the poor showing made by refraction by the manifest method and with eucatropine as compared to the results obtained with refraction with homatropine cannot be explained by the claim that the group of patients examined, having early presbyopia,

1. Mydriasis has little effect on the accommodation. As stated in a recent paper published in the ARCHIVES (An Evaluation of Homatropine-Benzedrine Cycloplegia, Arch. Ophth. **20**:585-596 [Oct.] 1938), I could find no change in the position of the punctum proximum in 13 of 15 cases after mydriasis with benzedrine sulfate.

had too great an accommodative power, for 61 per cent of the 100 patients required an addition greater than 1 diopter. The patients requiring an addition of more than 2 diopters exceeded those who required no addition by 3 per cent, and only 26 per cent required an addition of less than 1 diopter.

Still another approach to the study of the relation of refraction by the manifest method and with eucatropine to refraction with homatropine involves an analysis of the distribution among the several classes of persons with presbyopia (as presented in the preceding outline) of refractions obtained by the first two methods which are identical with those obtained with the use of homatropine:

	Manifest Refraction		Eucatropine Refraction	
	No.	%	No.	%
Class 1	..	...	2	7.7
Class 2	2	3.8	..	...
Class 3	5	5.5	7	7.7
Class 4	3	9.4	5	15.6

The occurrence of identical refractions in classes 1 and 2 is so rare as to make the percentage figure of little value. Class 3 gives a percentage accuracy for refraction by the manifest method of 5.5 and for refraction with eucatropine of 7.7. Class 4 shows a percentage accuracy for the manifest method of 9.4 and for refraction with eucatropine of 15.6.

Thus, there is established a definite trend toward greater accuracy with both manifest and eucatropine refraction the more advanced the presbyopia. It also appears that eucatropine refraction is somewhat more accurate than the manifest refraction and that this differential in accuracy increases with increasing depth of presbyopia. However, in class 4 (those persons requiring additions in excess of 2 diopters) absolute exactness in refraction as determined by comparison with homatropine refraction was attained in the case of the manifest examinations in only 9.4 per cent of the eyes refracted and in the case of eucatropine refraction in 15.6 per cent. This is true despite the common conception that the accommodation will not greatly interfere with the determination of the refraction by manifest methods after the age of 45. The percentages noted are high for the group as a whole, for class 4 constitutes only 16 per cent of the 100 patients. For the entire series of 200 eyes, the refraction with the manifest method showed a percentage of efficiency of 5 and with eucatropine a percentage of 7—rather too low to encourage the refractionist to dispense with cycloplegia in dealing with patients in this age group.

SUMMARY

A study is here reported of 100 patients from 40 through 50 years of age, refraction for each of whom was done by the same person by the fogging method of manifest refraction, by retinoscopic and by trial case examination after the instillation of eucatropine hydrochloride and by retinoscopic and by trial case examination after the instillation of homatropine hydrobromide, the latter being followed by postcycloplegic examination.

Eighteen per cent of the eyes examined by the manifest method and 12.5 per cent of those examined under the influence of eucatropine showed an increase in the amount of the cylinder over that detected by refraction with homatropine.

Decrease in the amount of the cylinder occurred in 23 per cent of eyes examined by the manifest method and in 22 per cent of those examined under the influence of eucatropine, the average amount of decrease being distinctly less with eucatropine.

Deviation of the cylinder axis occurred in 43.5 per cent of eyes examined by the manifest method and in 32 per cent of those examined under the influence of eucatropine, the average amount of deviation being greater with eucatropine.

Complete failure to detect cylindric error (evidenced either by failure to detect cylindric error which existed under refraction with homatropine or by detection of cylindric error the existence of which could not be confirmed by such refraction) occurred in 24.5 per cent of eyes examined by the manifest method and in 25 per cent of those examined under the influence of eucatropine. The amount of latent or false cylindric error was slightly less with eucatropine refraction than with the manifest method.

Comparison of the punctum proximum after manifest refraction with that after refraction with eucatropine demonstrated the development of cycloplegia in 64 per cent of the 72 eyes for which data were available.

In cases of advanced presbyopia, in which the addition required was in excess of 2 diopters, there was failure exactly to duplicate the homatropine refraction in 90.6 per cent of the eyes examined by the manifest method and in 84.4 per cent of the eyes refracted under the influence of eucatropine.

CONCLUSIONS

1. Refraction by the manifest method and with eucatropine does not compare favorably with refraction with homatropine because:

(*a*) There are sizable variations in the amount of cylindric error that each method uncovers.

(*b*) There is too frequent occurrence of variation in the location of the cylinder axis over a wide arc.

(*c*) There is complete failure to detect the cylindric error in a large proportion of the eyes examined.

2. Because of slightly less pronounced variations as enumerated, refraction with eucatropine appears to be slightly more efficient than with the manifest method.

3. Eucatropine is demonstrated to be a weakly cycloplegic as well as a mydriatic drug.

4. Refraction by the manifest method and with eucatropine, even in cases of advanced presbyopia, does not reveal the full cycloplegic refraction except for a small percentage of the eyes examined, the percentage being greater in the case of refraction with eucatropine.

5. All evidence indicates that for more optically precise refraction presbyopic patients in the fifth decade of life should be examined under homatropine cycloplegia rather than by the manifest method or after the use of eucatropine.

A NEW CORNEOSCLERAL SUTURE

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In 1894 Kalt [1] described the first corneoscleral suture. This consisted of two vertical bites, one in the cornea and one in the sclera, with 1 mm. between, in which the section was made. He later modified this procedure, inserting the scleral bite horizontally to make a T-shaped suture. Liégard [2] further modified Kalt's suture by placing both bites horizontally. This modification is often used today.[3] There are certain drawbacks to this type of corneoscleral suture. The wound is not covered by conjunctiva, although such protection may be partially obtained by a sliding Van Lint flap.[4] Instead of providing perfect approximation, the vertical arms of the suture allow a certain amount of lateral displacement, and because they run up over the lips of the wound they tend to produce inversion if tied too tightly. The latter fact may predispose to epithelial downgrowth as well as to imperfect union. Suarez de Mendoza [5] in 1892 tried to avoid some of these defects by making a preliminary incision from without in clear cornea and inserting the ends of a double-armed suture in either lip. Section was then completed through this prepared suture. While obviating some of the difficulties of Kalt's suture, this method had all the inherent drawbacks of a completely intracorneal section with unprotected edges and never became popular. Baldino [6] modified this method by inserting the needle, making the section up to it and completing the section by

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Kalt, E.: On the Corneal Suture in Cataract Extraction, Arch. Ophth. **23**:421, 1894.

2. Liégard, H.: Une modification au procédé de suture de la cornée dans l'opération de la cataracte, Ann. d'ocul. **149**:119, 1913.

3. (*a*) Ellett, E. C.: Use of the Suture in Extraction of Cataract, Arch. Ophth. **17**:523 (March) 1937. (*b*) Stallard, H. B.: A Corneo-Scleral Suture in Cataract Extraction: Its Technique and Advantages, Brit. J. Ophth. **22**:269, 1938.

4. Leech, V. M., and Sugar, H. S.: Reduction of Post-Operative Complications in Cataract Operations, Arch. Ophth. **21**:966 (June) 1939.

5. Suarez de Mendoza, F.: Nouveaux faits de suture de la cornée dans l'extraction de la cataracte, Bull. et mém. Soc. franç. d'opht. **10**:63, 1892.

6. Baldino, S.: Sutura corneale perfezionata, Rassegna ital. d'ottal. **4**:538. 1935.

dissecting out the needle and then pulling the suture through. Müller [7] in 1903 dissected a corneal pocket from below upward, placed two sutures in the edges of the pocket and then made a section up to the base of this corneal flap. Walker [8] in 1929 reversed this procedure, dissecting the corneal pocket from above downward. Verhoeff [9] in 1927 described two types of corneoscleral sutures with protection of the top of the wound by a sliding flap. The first suture was devised in an attempt to improve on Kalt's original suture by "sawing" a tract with the suture itself before making the section. The other suture was theoretically more perfect; a single vertical bite is taken at the limbus above, the section is made through it and the tract is then rethreaded after the eye is open. The last step presents obvious difficulties with unruly patients or when vitreous presents on section. Finally, in 1938 Lindner,[10] finding Verhoeff's sutures too difficult, reintroduced Suarez de Mendoza's method, placing the suture back in the sclera and using the hair of Japanese women instead of silk.

The ideal suture for closing cataract wounds should have certain definite characteristics. It should be inserted in solid corneal and scleral tissue and not in loose, yielding conjunctiva. It should be placed before the section is made and should not require extensive manipulations after the eye is opened. It should go through, not over, the lips of the wound, so as to give firm closure without danger of inversion of the edges, and it should be so placed that when tied it will bring the tissues back to exactly the preoperative position. The entire wound should be covered by conjunctiva as an added protection and better surgical closure. The method should not be too complicated for the average surgeon.

A suture which seems to meet these criteria has been developed at the Wilmer Ophthalmological Institute in the past year. The technic follows:

A small conjunctival flap is dissected down to the limbus around the entire upper half of the eye. At the base of this flap a small slot is made with a Lundsgaard knife (figs. 1 *A* and 2 *A*) about half-way through toward the anterior chamber. A cataract knife, keratome or small sharp scalpel could be used for the same purpose. A fine black silk suture on Kalt's corneal needle is then run through the base of the conjunctiva, reversed, and run through both the scleral

7. Müller, L.: Ein Operationsverfahren für komplizierte Stare und luxierte Linsen, Klin. Monatsbl. f. Augenh. **41**:11, 1903.

8. Walker, C. B.: Exactly Appositional Sutures in the Cataract Operation, Tr. Am. Ophth. Soc. **27**:51, 1929.

9. Verhoeff, F. H.: A Corneo-Sclero-Conjunctival Suture in Operations for Cataract, Tr. Am. Ophth. Soc. **25**:48, 1927.

10. Lindner, K.: Ueber Abanderungen der intracapsulären Staroperation, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **52**:392, 1938.

and the corneal lips emerging in clear cornea just below the base of the conjunctival flap (figs. 1 *B* and 2 *B*). The suture is thus placed so that when it is later tied it will bring the lips of the wound back to their original position before the eye is opened. The suture is then pulled out of the slot with a blunt iris hook (figs. 1 *C* and 2 *C*) and the loops laid aside to give room for the cataract knife. Section is made under the flap with a knife (fig. 1 *D*), which emerges at the base of the slot between the arms of the suture. Such a section is not as difficult as it sounds and has been successfully performed by inexperienced house officers on first trial. Extraction is performed in the usual way and the suture

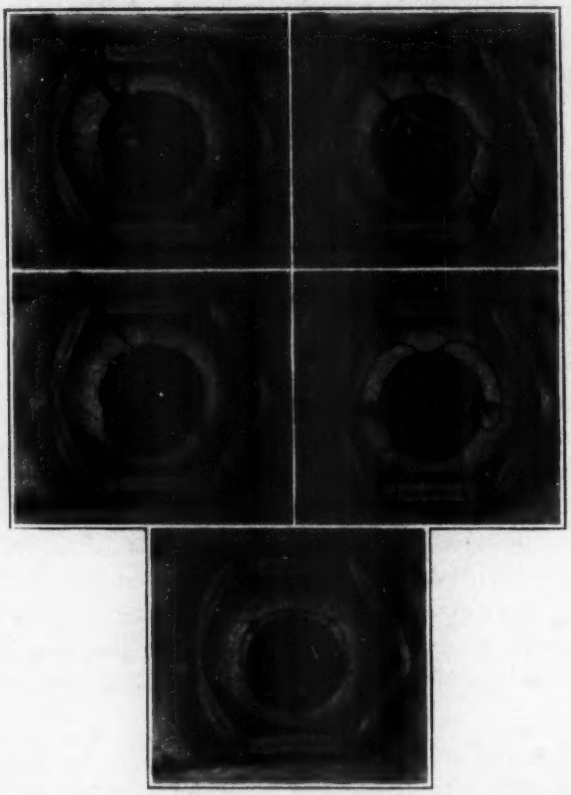

Fig. 1.—Technic of operation with the corneoscleral suture. In *A*, a small groove is made beneath the conjunctival flap with the Lundsgaard knife. In *B*, the suture is placed through the scleral and the corneal lips of the groove. In *C*, the suture is drawn out of the groove with a blunt iris hook. In *D*, section is made with a cataract knife, which emerges through the groove above. In *E*, the corneoscleral suture is tied and two additional conjunctival flap sutures are placed at the end of the operation.

pulled taut and tied at the end of operation (figs. 1 *E* and 2 *D*). If irrigation or extensive toilet of the wound is necessary, the suture may be tied before, making these maneuvers much safer. Further sutures may be placed in the edges of the conjunctival flap if this seems advisable to hold it in place, but firm closure of the wound is maintained by the corneoscleral suture. Although one such suture

gives excellent closure with perfect apposition, two, or even three, may be used when the operator is particularly apprehensive about a patient's postoperative behavior. The suture is not at all irritating and can be left in place for some time. Although Ellett [3a] advised removal of corneoscleral sutures on the third or fourth day, and Stallard [3b] on the fourteenth day, it has been the general practice in the institute to remove these sutures between the tenth and the twelfth day.

RESULTS

Ellett,[3a] Stallard,[3b] Lindner [10] and many others have pointed out the advantages of corneoscleral sutures: (1) firm closure with prompt healing, (2) reduction of postoperative hyphema, (3) less risk of

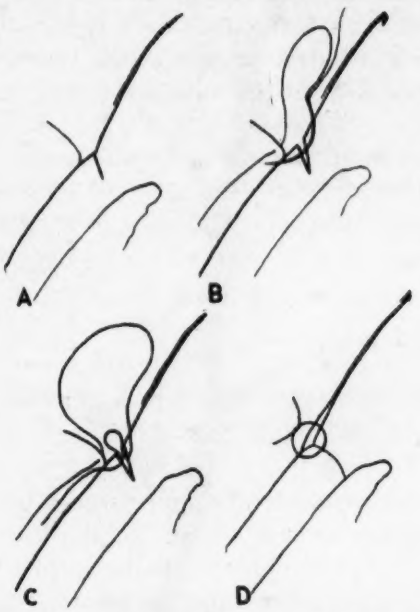

Fig. 2.—*A*, schematic cross section of the prepared slot behind the conjunctival flap. *B*, schematic cross section of the insertion of the corneoscleral suture. *D*, schematic cross section of the corneoscleral suture at the end of the operation. *C*, schematic cross section of the suture being drawn out of the groove.

prolapse of the iris or vitreous, (4) safer repair of prolapse of the iris if it should occur, (5) less postoperative astigmatism, (6) better closure if vitreous presents at operation, (7) earlier reformation of the anterior chamber and (8) greater freedom of movement for elderly patients and less danger for uncooperative ones.

Only Leech and Sugar have presented statistics to bear out these contentions. In their series of 450 cases, the incision was closed without a suture in 150, with conjunctival flap sutures in 150 and with a corneoscleral suture of a modified Liégard type in 150. Prolapse of the iris, vitreous or both and delayed reformation of the anterior chamber

occurred most frequently in the cases in which no suture was used and least frequently in those in which the corneoscleral suture was employed. The incidence of postoperative hyphema was least with the corneoscleral suture, but, interestingly enough, greatest with the conjunctival flap suture. This is probably a coincidence, but it might be a result of firm closure of the conjunctival wound with subsequent gaping of the unsupported corneoscleral incision beneath it under pressure of newly formed aqueous. The average amount of postoperative astigmatism was greatest when no suture was used and least when the corneoscleral suture was employed. These operations were all performed in the same institution but by various surgeons.

The series of cataract extractions here reported are those done by me during my year as resident surgeon at the Wilmer Ophthalmological Institute. They provide a smaller number of cases but a series in which

Comparison of Results with Both Types of Sutures

	Corneoscleral Suture	Conjunctival Suture
Total number of cases	110 (100%)	64 (100%)
Delayed reformation of anterior chamber or delayed closure of wound	4 (3.6%)	9 (14.1%)
Hemorrhage into anterior chamber	4 (3.6%)	12 (18.7%)
Choroidal hemorrhage	0	2 (3.1%)
Prolapse or incarceration of iris	3 (2.7%)	4 (6.3%)
Average cylinder	1.32	2.56

all operations were performed by the same surgeon under similar circumstances and with similar technic except for the use of either the conjunctival or the corneoscleral suture. Similar results have been obtained with this corneoscleral suture by other surgeons at the institute, but in order to keep all other factors constant only my results are analyzed.

In all, 174 extractions were performed. One hundred and twenty-nine were for ordinary senile cataracts and 45 for complicated cataracts of various types. Twenty-one were extracapsular extractions and 153 intracapsular extractions. In 99 cases one corneoscleral suture, as previously described, was used; in 11, two such sutures were used. In the remaining 64 cases closure was done with multiple conjunctival flap sutures. There are therefore available for comparison 110 cases in which the corneoscleral suture was used and 64 cases in which only the conjunctival suture was used. This comparison, with complications and the final amount of astigmatism, is summarized in the accompanying table.

Delayed union of the lips of the wound or delayed reformation of the anterior chamber was markedly reduced by the use of the corneo-

scleral suture (3.6 per cent contrasted with 14.1 per cent). The most striking improvement attributable to the use of this type of suture was the reduction of hemorrhage into the anterior chamber from 18.7 to 3.6 per cent. This complication was generally encountered around the fourth or fifth day; it was sometimes related to undue trauma on the part of the patient and more often appeared spontaneously, apparently without definite reason. All instances of seepage of blood into the anterior chamber were recorded, no matter how slight. Although the two deep choroidal hemorrhages occurred after the use of conjunctival flap sutures, this cannot be considered significant. Small incarcerations of the iris occurred in 3 cases (2.7 per cent) in which the corneoscleral suture was used, in 2 of them after extraction with round pupils, but none was severe enough to require repair. Prolapse or incarceration of the iris was found in 4 cases (6.3 per cent) in which conjunctival suture was used, in 3 of them after extractions with full iridectomy. One of these was only a minor incarceration, but the other three were true prolapses and were repaired. It is interesting to note that the average cylinder when the corneoscleral suture was used was almost exactly half that found when the conjunctival suture (1.32 diopters contrasted with 2.56 diopters) was employed.

SUMMARY

A new type of corneoscleral suture is described. Use of this suture has resulted in a marked reduction of gaping wounds, postoperative hyphema, prolapse of the iris and postoperative astigmatism.

THE ORTHOPTIC TECHNICIAN AS AN AID TO THE OPHTHALMOLOGIST

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The function of the orthoptic technician is to aid the ophthalmologist in establishing habits of correct binocular vision. In establishing any habit, the desired activity must first be physically possible for the patient; it must be within his capacity to perform the act. It is not always possible to determine with a single lesson whether the function of binocular vision is impossible or merely in abeyance. Once the possibility of binocular vision is established, however, the steps which follow are a typical educational process and constitute the second aspect of habit training.

The ophthalmologist may conveniently think of his patients for orthoptic training as falling into two classes: those on whom he expects to operate and those for whom he does not expect to use operative procedure.

For the first group he may describe preoperative and postoperative training. Preoperative orthoptics may begin with the treatment of amblyopia ex anopsia. Orthoptics may be made useful both by office treatments consisting of flashing and movement on orthoptic machines, monocular reading with graded print on the metronoscope and similar devices to stimulate the amblyopic eye, and by suggestions to the patient and family for home activities that will help maintain interest and cooperation through the often tedious period of reducing the amblyopia.

Regular training can begin as soon as the visual acuity is sufficient for the patient to see the targets, if necessary with especially favorable lighting, which may be brighter for the amblyopic eye.

The first thing which the orthoptist measures is anomalous correspondence. It is not within my province to define or explain this still moot condition, but to the technician it manifests itself on the synoptophore as a use of the eyes, which, if not corrected, makes subsequent training fail in its effect. It usually takes from ten to twenty lessons to establish normal correspondence, and once it is corrected it shows little tendency to relapse. Very often correcting the correspondence will result in a slightly improved position of the eyes.

If anomalous correspondence is not present, or after it has been corrected, the next step is the development of fusion with amplitude, if

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possible to include the position of balance while simple targets are being fused. Some patients lack fusion ability; they have no fusion sense. With the development of improved orthoptic instruments and technic, such persons seem to be fewer and fewer. Experienced technicians report that they have never seen a patient with true alternation; they all break down under training.

The first step in fusion training is to find out just the conditions necessary for fusion: the angle of fixation, the lighting and the type of target to which the patient responds the best. Patients often have idiosyncrasies regarding a target and will work better with one than with another of similar type. They will sometimes ask for hyperadjustments or even cycloadjustments, which are discarded as soon as training is begun.

A common obstacle to fusion is the crossing over, or suppression, of targets in whole or in part at just the point where fusion should take place. Such suppression acts like a scotoma and can be so located and mapped. Training will show a systematic reduction in the size of the scotoma. When the surrounding areas are successfully fused, the scotoma gradually disappears.

A second difficulty is extreme instability of fusion. The images fuse first in one position and then in another. The orthoptist watches the corneal reflexes and seizes favorable positions for stimulation until fusion becomes more marked within limited areas and there is a definite position from which to work.

Once fusion is established, the development of amplitude follows. It is usually somewhere at this point of the training that the ophthalmologist operates, if he plans to do so at all. The time element is often of importance, and the development of adequate amplitude may take several months of orthopic training alone or may be beyond the power of this technic in specific cases.

Postoperative training should begin as soon as the eye is sufficiently healed to make it safe, usually within a week or ten days. The muscles and coordination are then still pliable, and much can be accomplished in establishing a correct position and in overcoming former faulty habits of use. The development of amplitude is continued, with training in depth perception. Home exercises are taught, together with bar reading and other checks that will help the patient to understand how well his eyes are functioning.

Among nonoperative conditions, there are two special types that I wish to discuss: accommodative squints and convergence insufficiencies.

Accommodative squints may or may not belong in the nonoperative category. If a person has no more than about 3 diopters of hypermetropia and can begin training before 7 years of age, the prognosis for cure by orthoptics is favorable. There are different definitions of

cure of accommodative squints, but to the orthoptist it means binocular vision without deviation, muscle balance for near and far vision with the cover test, and four lights for distant vision and four or five for near vision with the Worth four dot test. To achieve this objective, it is necessary to teach the patient to dissociate his accommodation and convergence. When he learns that by seeing blurred images he can keep his eyes straight and still fuse, he has accomplished an important step. A person with mild hypermetropia will often then accommodate and obtain clear vision without converging again. It is this type of patient who can be encouraged to go without his glasses, at first for an hour or so at mealtimes, when he can be corrected readily if his eye deviates without his realizing it. Of course if he has much astigmatism or too high a degree of hypermetropia, he will not get clear vision in any case. This is one reason why such persons must be selected with care in order to obtain good results by orthoptics alone.

The sequence of treatment for such persons begins exactly as for those who are to be operated on: correction of amblyopia; correction of anomalous correspondence, which is especially common in accommodative squints, and development of fusion. As fusion training proceeds, it also teaches the patient to appreciate diplopia, first with targets and later with reference to casual seeing. The use of small lights, colored glass and similar devices stimulates appreciation of diplopia. Home exercises with lights and the finger help speed up this phase of training. It is important to lay a thorough groundwork at this stage, for it is by means of diplopia that the patient learns when his eye is deviated. The development of amplitude to balance gives him the power to fuse; the development of diplopia gives him the key to maintaining fusion.

Experience has proved that the diplopia that appears as a stage of orthoptic training is not nearly so distressing as other types of diplopia. It is fleeting, tends to be vague and soon disappears in favor of fusion.

It takes from four to eight months to teach an intelligent child this correction of accommodative squint, and the results are usually gratifying. Treatment may be discontinued at any point that the results seem adequate to meet the situation, or it can be carried on until complete control is developed. In no case can worth while results be expected in less than three months with two or three treatments a week, and if it is unlikely that the patient will cooperate for this length of time it is not hopeful that success can be achieved by orthoptics alone.

Convergence insufficiencies yield well to orthoptic training. The typical subjects are young adults who train and cooperate well. They are usually willing to do home exercises as a supplement to office training. They develop amplitude rapidly, and when their margin of reserve

is adequate they find convergence easy and comfortable. Two to six weeks brings striking improvement in such cases.

Since orthoptics cannot correct anatomic anomalies, either congenital or acquired, it is of the greatest importance that the causation of the condition for which training is given shall be clear so that patients suitable for training are selected. It is often impossible to give a satisfactory prognosis until after two weeks of treatment, or even longer in difficult borderline cases.

Orthoptics is essentially a process of reeducation, subject to the limitations of educational procedure.

490 Post Street.

LINDAU'S DISEASE

REPORT OF SIX CASES, WITH SURGICAL VERIFICATION IN FOUR LIVING PATIENTS

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In 3 cases a diagnosis of Lindau's disease [1] has been verified recently by craniotomy at the Toronto General Hospital, and in a fourth case, that of a blind sister of 1 of the patients in the aforementioned 3 cases, a cerebellar hemangioendothelioma was removed in Vancouver, B. C., in 1938. All 4 patients have since returned to their work. The mother in one of the two families involved died after an operation for tumor of the brain in 1918; the father in the other family died in 1916 of intracranial complications; both of them had been blind in one eye for five years before death. In all, 6 of the 11 members in two generations have been affected, and the surgical diagnosis has been confirmed by pathologic examination in 2 of the cases in which the patients are living.

Before 1928 fatal results were uniformly reported in the treatment of Lindau's disease. The earliest records have shown clearly the familial incidence of multiple angiomas involving the central nervous system, principally the cerebellum. The cerebrum, the spinal cord and the retina are of secondary importance to the cerebellum as sites for the angiomas that accompany Lindau's disease, according to statistics on fatal cases. Of less importance are cystic changes in kidneys, pancreas, epididymis, liver and bones and the finding of another variable factor, hypernephroma. It is to be noted that the fibrovascular layer of the retina was the site of Hippel's disease, or angiomatosis retinae. This layer is embryologically allied with brain tissue.

The oldest patient is reported as being 48 years of age,[1a] but the condition is usually seen in the second or the third decade of life. It is slowly progressive, and in recent years in a few verified cases it has, it is hoped, been permanently arrested by extirpation, or when gross removal was impossible on account of the danger of damage to surrounding structures, by decompression, local diathermy and tapping, with the addition of irradiation.

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society at Hot Springs, Va., June 6, 1939.

1. Lindau, A.: Acta ophth. 4:193, 1927.

1a. The data on this patient are included in the condensed table of cases of Lindau's disease with surgical verification in living patients.

Dr. K. G. McKenzie, of the neurosurgical department of the Toronto General Hospital, surgically verified the diagnosis of Lindau's disease in 3 of these cases. The blind sister of 1 of the patients was able to resume her work at the Institute for the Blind after an operation in Vancouver, B. C., Canada, when a cerebellar hemangioendothelioma was removed by Dr. Turnbull [2] in 1938. In a series of 53 verified cerebellar tumors at the Toronto General Hospital there were 10 other cerebellar hemangiomas (1932-1938 inclusive); these did not have a hereditary relation with Lindau's or Hippel's disease.

The year 1928 may also mark the division of Lindau's disease into two categories, fatal and nonfatal, as it is evident that the widespread complications reported by the early writers will no longer be seen. The ophthalmologist is principally concerned with the second category, in which there is hope for recovery. The cerebellar tumors of Lindau's disease are found in about 1 of every 5 patients who suffer from angiomatosis retinae, or Hippel's disease. Angiomatosis retinae also has a familial incidence according to Usher,[3] who in 1936 collected 119 cases, in 18.48 per cent of which the condition was inherited, 22 patients in 8 pedigrees being affected.

A review of the records of the complete syndrome of angiomatosis retinae plus cerebellar tumor shows less than 50 cases, and it may be doubted that the condition in those reported by Isnel,[4] Davidoff,[5] Wessely [6] and others should be considered Lindau's disease, as a factor of heredity, angiomatosis retinae or cerebellar tumor is wanting; in other words, hemangioendothelioma alone is not Lindau's disease.

The cases of Lindau's disease in the second family reported on here may be considered as belonging to the list of cases of Lindau's disease in which diagnosis is yet to be confirmed. There is no record in case 4 of a retinal angioma, and the cerebellar angioma was lost at operation, when the small mural tumor disappeared in the sucker as the surrounding cyst was evacuated. The sister of the patient was completely blind before a hemangioendothelioma was removed from her cerebellum, and the father died of an intracranial complication after five years of blindness.

The failure to diagnose the condition in case 1 as Lindau's disease until after the sister's operation leads me to believe that blindness without a history of trauma, when associated with the factor of heredity,

2. Turnbull, F.: Personal communication to Dr. K. G. McKenzie.
3. Usher, C. H.: Tr. Ophth. Soc. U. Kingdom **50**:183, 1935.
4. Isnel, R.: Bull. Soc. d'opht. de Paris, December 1934, p. 652.
5. Davidoff, L. M.: Am. J. Path. **5**:141, 1929.
6. Wessely: Klin. Monatsbl. f. Augenh. **90**:95, 1933.

should be considered, for the purpose of diagnosis, as a possible equivalent of angiomatosis retinae. The essential cause of blindness in case 1 was obscure until the sister's operation, and even then confirmation was difficult by pathologic examination on account of the small size of the retinal tumor.

The successful treatment of angiomatosis retinae by radon seeds has been recorded by Foster Moore,[7] although a large defect of the visual field resulted. It is hoped that secondary glaucoma and retinal

Data on Ten Cases of Lindau's Disease with Surgical Verification in Living Patients

Reporter	Patient	Sex	Age	Heredity	Angiomatosis Retinae	Comment and Findings
Cushing and Bailey: Arch. Ophth. 57 : 447, 1928; Tumors Arising from the Blood Vessels of the Brain, Springfield, Ill., Charles C. Thomas, Publisher, 1928	F. McA.	M	30	+	Yes	Cerebellar hemangioma
Møller: Acta ophth. 7 : 244, 1930	C. C. H. J.	M	44	+	Yes; enucleation	Hyperplastic cerebellar capillary hemangioma
Viets: J. Nerv. & Ment. Dis. 77 : 457, 1933	F. L. W.	M	48	No	Yes	Inoperable cerebellar tumor; cure with irradiation
Atkinson: Arch. Ophth. 7 : 510 (April) 1932	J. R.	M	32	No	Yes	Angioma and cyst removed
Hope-Robertson: Australian & New Zealand J. Surg. 4 : 55, 1934	C. F. D.	F	16	+(?)	Detachment	Hemangioendothelioma; treated by diathermy
MacDonald	Miss R.	F	23	+	Yes; blind	Cerebellar tumor; irradiation; eye enucleated
MacDonald	Mrs. S.	F	20	+	Yes	Hemangioendothelioma of cerebellum
MacDonald	A. P.	M	30	+	No	Cerebellar tumor and cyst; specimen lost
MacDonald	Mrs. T.	F	29	+	Blind	Patient operated on in Vancouver; cerebellar angioma
Casten: Personal communication to author; Am. J. Ophth. 16 : 999, 1933	.....	M	48	No	Yes	Retinal cyst and pontile angioma

detachment will be prevented by radiation of a degree intermediate to the reported extremes. Therefore, in treating the patient in case 2 four radon seeds were used, each of 0.7 millicurie, attached to a silk suture at intervals of about 4 mm., for seven days on the sclera opposite the tumor.

The terms Hippel's disease and Lindau's disease must be retained in describing conditions associated with the names of Hippel and Lindau, for while both conditions have a familial tendency, Hippel's disease may occur without complications. The neurosurgeon and the

7. Moore, R. F.: Tr. Ophth. Soc. U. Kingdom **53**:215, 1933.

pathologist are more interested in syringomyelia, syringobulbia and cerebral cysts, which, in the absence of angiomatosis retinae, form part of the syndrome of Lindau's disease and have been reviewed by Wolf and Wilens,[8] Brandt,[9] Dandy,[10] Sargent and Greenfield.[11] The cystic changes in the pancreas and the kidney are of interest chiefly to the pathologist.

REPORT OF CASES

Family R.—CASE 1.—Miss E. R., a white woman aged 23, a nurse, was referred to Dr. K. G. McKenzie on Feb. 3, 1936, by Dr. William S. Butler, of North Bay, Canada. She had suffered from headache in the region of the vertex and vomiting for one year, which was increasing in severity. Two years previously, on bending over, she had suddenly lost the sight of her right eye, and when she was seen shortly after this by Dr. C. Hill it was stated that the loss of vision was due to an intraocular hemorrhage. Her appendix had been removed on account of the vomiting. A tentative diagnosis of tumor of the brain was made, and she was admitted to the Toronto General Hospital on February 4.

Inquiry as to past illnesses and functional factors revealed nothing of importance. Physical examination gave essentially negative results, showing only a healed scar at McBurney's point and blood pressure of 90 systolic and 60 diastolic. Neurologic examination showed good intelligence, cooperation and memory. Vision in the right eye was 6/18; with glasses it improved to 6/6. There was no perception of light in the left eye. The visual field of the right eye was normal; the physiologic cup was filled. There was slight blurring of the borders of the disk. The right pupil reacted to light and in accommodation. The left pupil was irregular and showed ectropion of the uveal pigment with a dense opacity in the lens. The fifth, eighth, ninth, tenth, eleventh and twelfth nerves were normal. The patient was right handed, and the strength in each hand was good. Sensation to touch, light, heat and cold was normal. The reflexes were normal, except that the triceps reflex was not obtained. Stance was good, although the patient felt weak. Romberg's sign was negative. Slight staggering on walking was possibly due to weakness.

A provisional diagnosis was made of congenital aneurysm about the circle of Willis.

On Feb. 10, 1936, Dr. McKenzie did a first stage cerebellar exploration. The patient reacted poorly, and the pulse rate rose to 150. The arch of the atlas was removed.

On February 11 there was no vomiting, and the right disk was still swollen.

On February 24 Dr. McKenzie performed a second stage cerebellar operation. The patient again reacted poorly, but a vascular tumor, about the size of a large walnut, was found when the dura was opened. It occupied the lower half of the right cerebellar hemisphere and caused direct pressure on the medulla. A large venous channel, ⅛ inch (0.3 cm.) in diameter, passed from the tumor to the midline to empty into the transverse sinus. It seemed impossible to remove the vascular tumor, so only decompression was done.

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8. Wolf, A., and Wilens, S. L.: Am. J. Path. **10**:545, 1934.

9. Brandt, R.: Arch. f. Ophth. **106**:127, 1921.

10. Dandy, W. E.: Arteriovenous Aneurysm of Brain, Arch. Surg. **17**:190 (Aug.) 1928.

11. Sargent, P., and Greenfield, J. G.: Brit. J. Surg. **17**:84, 1929. Paton, L., and Williamson-Noble, F. A.: Tr. Internat. Ophth. Cong. **2**:624, 1929.

By February 29 the patient had made a good recovery; there were no headaches or vomiting.

Beginning on April 9 sixteen irradiation treatments were carried out before the patient was discharged from the hospital.

Up to May 15 recovery had been steady. The margins of the right disk were still blurred.

On May 22 the patient was readmitted to the Toronto General Hospital and discharged on June 6. She had lost considerable weight and most of her hair had fallen out over the posterior three quarters of the scalp. She recovered considerably with rest in bed; there was no vomiting or headache.

On May 27, 1937, when I first saw the patient, vision was 6/18 + 1 in the right eye; there was no perception of light in the left. The pupil in the right eye was active, but the left was fixed and irregular, the iris being bound down by a complete posterior synechia. There was some ectropion of the uveal pigment. There was a dense opacity in the lens, and no fundus reflex could be seen. The right eye showed clear media, the borders of the disk were normal and there was slight pulsation in the vein and a few flecks of pigment in the foveola. The tension in the left eye with the Schiötz tonometer was over 80 mm. Slit lamp examination of the left eye showed a cloudy cornea, a shallow anterior chamber and vessels close to the posterior surface of the lens. Transillumination of the left eye seemed to indicate that a tumor was present, up and in. Enucleation was advised, but in the hope of being able to save the eye the patient was admitted to the hospital on March 30 for extraction of a cataract with a full iridectomy. The eye remained irritable, and the patient returned on May 30. At this time there was marked pain, pericorneal injection and a drawn pupil. Enucleation was again advised, and on May 31 the left eye was removed, local anesthesia being employed. The patient was discharged from the hospital on June 4. The pathologic report showed secondary glaucoma, secondary cataract, detachment of the retina and calcification of the choroid.

Further examination of this specimen was undertaken only after the sister's condition was verified on May 6, 1938.

Pathologic Examination of Right Eye (figs. 1 and 2).—The corneal epithelium was intact. The cornea was vascularized beyond the limbus. The anterior chamber showed complete obstruction of the filtration angles, and even in the central region the iris was firmly bound to the cornea. The ciliary bodies showed a moderate amount of free hemorrhage. The retina was extensively detached in the posterior part, and the choroid showed large deposits of pigment and exudate in which calcium salts had been deposited. On one side near the optic nerve there was an extensive mass of acellular exudate containing clumps of pigment. Heavy masses of calcium were seen close by and also numerous clefts, where apparently cholesterol crystals had been dissolved out in the process of preparation. Much partly absorbed hemorrhage was seen at the ora serrata, invaded by a light connective tissue. A small tumor, measuring 2.5 by 3 mm., was seen in the anterior portion of the retina. Large vessels were present at the side of the tumor. The tumor itself showed many fine vessels lined with a single layer of endothelium and numerous partly formed vessels. Special stains, iron hematoxylin and van Gieson's stain, showed the connective tissue surrounding the larger vessels. The main mass of tumor contained little connective tissue but numerous fine capillaries. In places clumps of endothelial cells suggested the process of formation of new vessels. There was a moderate amount of general mononuclear infiltration surrounding the area of the tumor and of light connective tissue that extended forward to the ciliary

Fig. 1 (case 1).—Low power photomicrograph of a hemangioendothelioma in the anterior portion of the retina behind the ciliary body. The cysts were lined with endothelium.

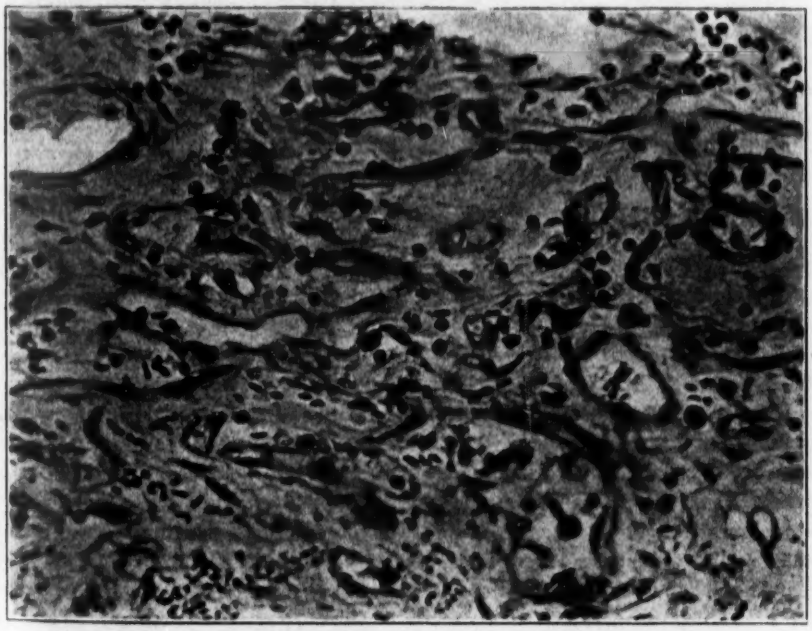

Fig. 2 (case 1).—High power photomicrograph (× 165) showing the detail of the angioplastic tumor in figure 1.

bodies, which were not detached. Large cysts lined by endothelium were seen close to the tumor in the anterior portion of the retina.

A diagnosis was made of retinal hemangioendothelioma.

Additional Data.—On Aug. 7, 1939 the patient was readmitted to the hospital because of vomiting and headaches and inability to work. On August 10 a cerebellar tumor on the left side was removed by Dr. McKenzie, with difficulty on account of large vessels. On August 26 the patient was discharged after a good recovery. On November 8 histologic examination of the tumor showed small endothelium-lined spaces containing blood. These spaces were separated by proliferation of endothelial cells. The tumor presented the typical appearance of a hemangioendothelioma.

CASE 2.—Mrs. L. C. S., aged 29, a sister of the patient in case 1, consulted me on May 30, 1937. She had noticed floaters in the right eye for three years and had worn glasses for one year. No headaches had been experienced, but the eyes tired easily, and she had noticed loss of sight in the right eye. The vision in the right eye was 6/60 and in the left eye 6/9. The pupils were equal and showed direct light, consensual and convergence reflexes. Examination without a mydriatic showed in the right eye a large opacity of the vitreous with a strand attached to it. The fundus showed numerous irregular whitish flecks, which coalesced about the foveola. They were widely scattered, but decreased in size in the region of the upper and lower temporal vessels. The veins were full and markedly large, and there was a pulsation in the vein at the disk. The flecks were considered to be due to degenerative changes. The fundus of the left eye showed clear media and a normal cup; a slight temporal crescent was present with an occasional fleck near the disk; the foveola was clear. The vision of the right eye did not improve with glasses, but that of the left eye improved to normal. The patient was referred to her physician for a thorough physical examination.

On October 25 she complained of loss of vision in the right eye and stated that she had noticed occasional blurring in the left eye. The vision in the right eye was limited to ability to count fingers at 1 meter; that in the left eye was normal with correction. Examination of the fundus revealed only a slight increase of the condition previously described about the foveola. The patient was referred to Dr. McKenzie, as she staggered at times and stated that no libido was present.

On March 4 she was admitted to the Toronto General Hospital to the service of Dr. McKenzie. She stated that she had been well until October 1937, when she had severe frontal headaches, loss of vision in the right eye and vomiting. She had consulted Dr. McKenzie on March 2, but at that time Dr. McKenzie did not feel that any surgical procedure was necessary, and she had returned home.

On May 6 the field of the right eye showed a cut to 40 degrees below the nasal meridian. The fundus of the left eye showed blurred borders. A small hemorrhage was seen in the left eye well out at the periphery, near a small globular whitish mass. There was no history of past illnesses. The patient weighed 112 pounds (50.8 Kg.), having lost 23 pounds (10.4 Kg.) during the previous two months. Physical examination gave essentially negative results. Neurologic examination showed the third to the twelfth nerves to be normal. Examination of the eyes gave essentially the same results as on May 30. With the Romberg test the patient had a tendency to fall to the right, and she staggered to the right on walking. There was no paralysis or wasting.

A provisional diagnosis of cerebellar tumor on the right side, probably Lindau's disease, was made.

On May 16 Dr. McKenzie did a cerebellar exposure and found in the upper part of the left hemisphere a vascular lesion attached to the dura, lateral sinus and tentorium by a large, thin-walled vessel. Hemorrhage was free, and a tumor the size of a large walnut was removed. It shrank considerably on fixation and showed a yellow cyst, ¾ inch (1.9 cm.) in diameter, filled with yellowish fluid and lined by a pinkish smooth surface (fig. 3).

Pathologic Examination.—Tissue from the part of the tumor which is marked with a cross in figure 3 was examined and a diagnosis of hemangioendothelioma of the cerebellum made. The tumor tissue consisted of small vascular spaces separated by masses of endothelial cells, which were foamy in appearance. The silver stain showed a well marked network of reticulum fibers.

Ophthalmoscopic Examination.—On May 17 the right eye was dilated. There was a large floating mass well forward on the vitreous; above this was a strand that increased in thickness as it passed up and out to a mass at the periphery.

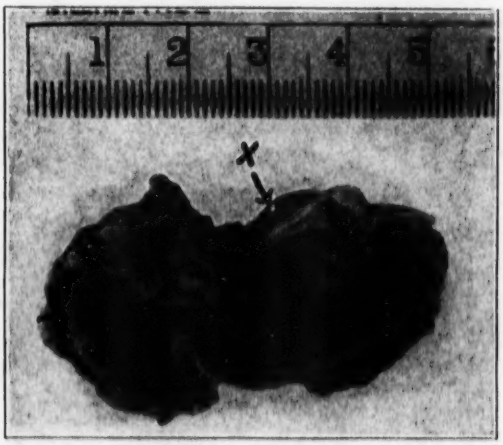

Fig. 3 (case 2).—Gross specimen of a cerebellar hemangioendothelioma. The section in figure 4 is from the area indicated by the cross.

With a + 12.00 sphere vessels were seen on this mass. A + 15.00 sphere showed only white connective tissue; some pigment was seen near the entrance of the greatly dilated vein and the artery. There was a veil of connective tissue over the disk, which ended in a knob seen with a + 5.00 sphere. The borders of the disk were blurred, especially above, below and nasally. On the temporal side heavy yellowish white areas with brilliant flecks were seen extending from the upper temporal to the lower temporal vessels; they were dense in the foveolar area. Patches of these multiple flecks decreased in number to the temporal side, and finally only faint discrete flecks were seen. Fine pigment (blood staining possibly) was seen along the lower temporal vessels, well out. The upper temporal artery was irregular, and where it passed over a white area the sharp borders presented slight bulgings, so that the caliber of the vessel was variable. This was more evident toward the periphery. The upper temporal vessels converged and passed into the aforementioned mass, which looked white in the central part and rather grayish at the edge. One small dilated capillary, or light hemorrhage, was seen at the lower border.

The media of the left eye were clear; the borders of the disk were slightly blurred above, below and on the nasal side, and the veins were dilated. There was one small flame-shaped hemorrhage below at the border of the disk and another small one above. There was a marked pulsation in the vein, while there was only slight pulsation in the right eye. Outside of an irregular light reflex, the foveolar region showed no change.

Course.—On May 20, 1939, the patient complained of further loss of vision. Her general condition was good. The fundus of the right eye showed a flat detachment near the tumor, with dark vessels, so that the artery and vein seemed alike unless followed from the disk. The central exudate and also the number of brilliant flecks had increased. The beading was more marked. Four 0.7 millicurie radon seeds were inserted opposite the tumor, to give a total dose of 2.8

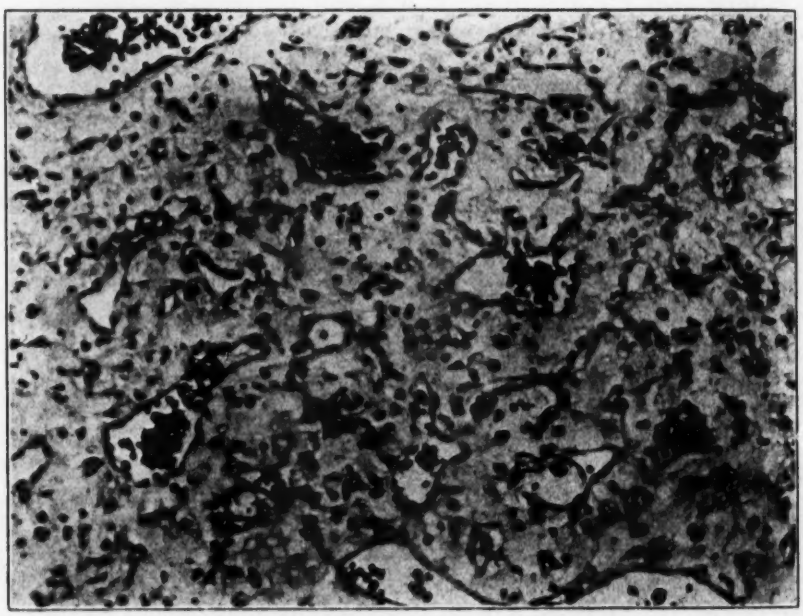

Fig. 4 (case 2).—High power photomicrograph (× 85) showing the detail of the angioplastic cerebellar tumor.

millicuries for seven days. The removal was facilitated by dipping the seeds tied to a silk suture in liquid pyroxylin before insertion.

Case 3.—Mrs. John R., the mother of the patients in cases 1 and 2, consulted Dr. Minnes, of Ottawa, Canada, in 1912, at the age of 22, according to her daughters, from whom an incomplete history was obtained. Dr. Minnes' notes, supplied by Dr. Wilbur Fraser, of Ottawa, stated that on July 2, 1912, the right eye had been sore for eleven years, following measles. It had been frequently painful, and this severe attack had lasted for one week, but sight had been wanting for over five years. A complete annular synechia was present, with opacities in the lens, and there was no perception of light. On July 3, 1912, enucleation was performed, and when the globe was opened it was found that there was complete ossification of the choroid, so that a bony cup was formed. A search was made

by Dr. Hooper in Dr. Webster's notes and also in the records of the Civic Hospital, but no report of an operation on the brain could be found, although the daughters stated that their mother died at the age of 32 after an operation on the brain for a suspected tumor.

Family P.—CASE 4.—Albert P., a white man aged 30, a gardener, was admitted to the Toronto General Hospital on Oct. 19, 1937. His chief complaints were attacks of dizziness and nausea, occasionally accompanied by headache. He had felt well until fourteen weeks before the present illness, when he suffered from his first attack of dizziness and nausea. A second attack followed three days later. Severe headaches would start at any time, even at 3 a. m., and last the whole day or at times only a part day. Sight was blurred in the last three weeks before admission, especially in the right eye. The patient tended to stagger when walking. There were no past illnesses, and inquiry into the personal history elicited nothing of importance. The family history showed that the father had become blind at 35 and died from cerebral complications at the age of 40. One sister had become almost blind during the past few months. One brother was alive and well.

Functional examination gave essentially negative results. The patient's memory was good.

Physical examination gave essentially negative results. The reflexes were hyperactive but equal throughout.

A differential diagnosis of an intracranial tumor or neurosis was made, with a provisional diagnosis of intracerebellar tumor on the right side.

On November 3 there was a fine nystagmus, and the eyes tended to drift to the right. The ophthalmoscope showed a small recent hemorrhage near the disk at 2 o'clock, in the right eye. There was loss of physiologic cupping; engorgement of the vein was present, and the margins of the disk were blurred.

On November 5 cerebellar decompression was done by Dr. McKenzie with the patient under ether administered endotracheally. A cyst containing yellow fluid was encountered in the midline of the cerebellum. A pink tumor, about 1 cm. in diameter, lying on the deep wall of the cyst, was picked up with the sucker and disappeared with the contents of the cyst, so that no tissue was obtained for pathologic examination.

The patient made a fairly satisfactory recovery and was discharged on December 24. He was able to stand with his heels together and his eyes closed; some nystagmus was still present, but the vomiting that he had experienced before the operation, especially if he turned on his back or to the right side, had disappeared.

By Jan. 21, 1938, the patient was greatly improved; there was still some dizziness, but the nystagmus was less.

By February 25 the patient was not able to read for any length of time, and he became dizzy with any exertion.

By July 17 he had returned to his former occupation, free from headaches and staggering.

Laboratory examination showed a negative Wassermann reaction, a cerebrospinal fluid pressure of 230 mm. and a blood pressure of 105 systolic and 75 diastolic. Otherwise the results of the examination were negative.

CASE 5.—On Dec. 28, 1938, Dr. Frank Turnbull, of Vancouver, B. C., wrote to Dr. McKenzie, stating that he had operated on Mrs. C. T., aged 29, a sister of the patient in case 4, six months previously for a cerebellar tumor of the hemangiomatous cystic type. He stated that she had been totally blind for several months with bilateral retinal detachment. Dr. Turvey suggested that the condition

was Lindau's disease. The patient made an excellent recovery after the operation and resumed her occupation at the Institute for the Blind. It was confirmed that the blind father had suffered from severe headaches and died in 1916.

CASE 6.—The father of the patients in cases 4 and 5 became blind at 35 years of age. He died at about 40 years of age from complications of an intracranial nature. No record has been found for him.

COMMENT

It will be seen that in two generations of 2 families 6 cases of Lindau's disease have been found among 11 members. Of the remaining 5, only 1, Mrs. D., a sister of Miss R. and Mrs. S., was seen on July 16, 1938, at the age of 28. She consulted me as to whether a tinted + 0.50 sphere which she had been using for each eye was necessary. Examination showed vision of 6/6 — 2 in each eye. The pupils were large and equal and showed direct light, consensual and convergence reflexes. The bulbar conjunctiva of the left eye showed a diffuse nevus 3 by 3 mm. near the limbus at 9 o'clock; two large vessels passed from this area to below the caruncle. The fields of the right and left eyes were full, and the patient recognized 1 mm. red, blue and green test objects with each eye. The fundi showed clear media and a pulsation in the vein on the disk, more in the left than in the right eye. In the left eye slight tortuosity of the vessels was noted. With correction of a + 0.50 cylinder in each eye at 90 degrees vision was normal. A drop of an astringent solution was ordered for the slight redness that followed reading.

Mr. R., brother of the patients in cases 1 and 2, a school teacher, was not examined. His vision was said to be normal and his health good.

In family P. it was said that the unexamined brother had normal sight. The 2 unaffected members of the first generation, the father in family R. and the mother in family P. were said to have had normal sight.

CONCLUSIONS

1. The successful treatment of Lindau's disease seems possible, but further observation of treated patients is needed to show if the apparent relief of symptoms is lasting.

2. The ocular tumor may easily be missed at pathologic, as well as at clinical, examination. It was a small peripheral notch in the nasal field of 1 of my patients that led to the discovery of the tumor.

3. The possibility of angiomatosis retinae as a cause for blindness in the second and third decades must be borne in mind, for secondary glaucoma or hemorrhage may obscure the diagnosis.

4. Angiomatosis retinae occurs in the cerebral layer of the retina and may be one of multiple hemangioendotheliomas of the central nervous system.

5. Hemangioendothelioma is susceptible to treatment by irradiation.

6. Multiple hemangioendotheliomas must be demonstrated to justify a diagnosis of Lindau's disease, or if only one tumor is found pathologically, familial incidence must be shown.

7. A sister of the patients in cases 1 and 2 had a conjunctival nevus.

8. Both terms, Lindau and Hippel disease, must be retained.

DISCUSSION

DR. ALLEN GREENWOOD, Boston: I wish to mention a case which had been extremely interesting to me, that of a schoolteacher of 28, whom I presented at a meeting of the New England Ophthalmological Society some twelve years ago. I presented her because her fundus was easily seen and had two enormous veins in it, one running straight up and one running straight down. They were three or four times the size of the normal vein, and each one ended in a clear, round, shining cyst; a diagnosis of hemangioma was made. The patient, by the way, had a cousin who had had the same condition. The patient had lost her other eye because of separation of the retina and glaucoma. The eye was removed by Dr. Standish, and no examination was made, as far as I can find out. I sent the patient immediately to Dr. Harvey Cushing at the Peter Bent Brigham Hospital with the diagnosis I had made, to see if he could find any evidence of involvement of the cerebellum. He found none, but he had his artist make some beautiful pictures of the two cysts, which later became obstructed by disturbance in the vitreous. Dr. Cushing had roentgen irradiation used to see if it would stop the progress of the cysts and prevent blindness. A few years later the patient was led into my office by her uncle; she was led in because she was so dizzy she could not walk properly. She was sent again to the Peter Bent Brigham Hospital. Dr. Cushing had left, but Dr. Cutler made a diagnosis of cyst of the cerebellum, which he removed, and the last I knew of the patient she was living but with very poor vision. The cousin had been blind in both eyes, due to what was probably the same condition. I have not heard from this patient for several years, but, as I say, the last I knew she was still living, having had a tumor removed from the cerebellum. This was unquestionably a case of so-called Lindau's disease.

DR. WALTER S. ATKINSON, Watertown, N. Y.: Dr. MacDonald has referred to a case of Lindau's disease that I reported in 1932, and I wish to have recorded that this patient is still alive and well, although his gait is a bit unsteady. He has had two recurrences of the cerebellar cystic angiomatous tumor and has had two more operations, three in all. The eye with the angiomatosis retinae is blind due to total detachment of the retina. The other eye is not affected, and the vision is good.

DR. HENRY P. WAGENER, Rochester, Minn.: As MacDonald has stated, the diagnosis of a complete Lindau syndrome cannot be made

before the patient comes to necropsy, because one cannot demonstrate the presence of the other congenital anomalies which Lindau made a part of his complete syndrome. However, I do feel that one is justified in making a diagnosis of Lindau's disease when there is present a definite angiomatosis of the retina in association with a surgically proved hemangioma of the cerebellum. Craig, Kernohan and myself are reporting at the American Neurological Society 4 cases, in 1 of which necropsy was performed and the complete syndrome was confirmed, the presence of the angioma in the retina in each eye being confirmed histologically, and in the 3 of which confirmation was made surgically and the patients are still alive so far as we know.

SYNDROME OF NEUROMYELITIS OPTICA

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So complex is the nervous system and so manifold its functions that one and the same etiologic agent may produce many different syndromes by the pure chance of the site and the tempo of involvement. A disease affecting the upper or lower lobe of the lung, some portion of the liver or the right or left kidney may give similar symptoms and is considered under one designation. However, if there is a virus infection in the nervous system and it happens to attack the second cranial nerve, it may be called "papillitis"; if the vestibular portion of the eighth nerve is involved, one reads of "epidemic vertigo"; if the seventh nerve is invaded, there is facial paralysis.

As the possible combinations of involvement of the brain, cranial nerves, brain stem and spinal cord are numerous, so too are the designations or the syndromes.[1] These syndromes have a certain historical significance. They constitute fine descriptions of disease processes, but there is a tendency to confusion and an impression that many distinct and widely different disease processes are being dealt with. For instance, one reads in the French literature such terms as neuritis, neuronitis, plexitis, schwannitis, ganglionitis and many combinations involving the spinal cord.

Such titles are useful as clinical guides if it is understood that each may represent merely a phase or subdivision of a major group and not a distinct disease.

In the past there has been a tendency to subdivide the entire group of nonsuppurative infections of the nervous system into four major concepts. According to Brain [2] and Berliner,[3] these include multiple sclerosis, Schilder's disease, acute disseminated encephalomy-

1. Strauss, I.; Rabiner, A. M., and Ginsburg, S. W.: The Clinical, Bacteriological and Epidemiological Aspects of Encephalomyelitis, A. Research Nerv. & Ment. Dis., Proc. **12**:262, 1932. This article contains different points of view on this subject and an extensive bibliography.

2. Brain, W. R.: Critical Review: Disseminated Sclerosis, Quart. J. Med. **23**:343, 1930.

3. Berliner, M. L.: Acute Optic Neuritis in Demyelinating Diseases of the Nervous System, Arch. Ophth. **13**:83 (Jan.) 1935.

elitis and neuromyelitis optica. Even this subdivision is open to criticism, because there are many neurologists who believe that these designations apply to the same disease in its different manifestations. There are many able neuropathologists, among them Hassin,[4] who make a fundamental distinction between these various disease processes.

There is need for a unified terminology, which it is hoped will be forthcoming when the etiologic agent can be better identified. At the present time, the majority of authors agree with Rivers [5] that the cause of such illnesses is a virus and that the clinical course of the disease is subject to marked variations.

Among the combinations of areas affected, which may turn up with remarkable frequency (like 7 and 11 on dice), are the optic nerve and the spinal cord. This combination is well known to every ophthalmologist and to neurologists, who, if they encounter a case of retrobulbar optic neuritis, look for and expect to find evidence of pathologic changes in the spinal cord at the same time or at a later date. When the involvement of the combined areas is acute and striking, it has been called neuromyelitis optica. The earlier case reports which called attention to this association were those published by Albutt [6] in 1870 and by Erb in 1879.[7] Devic [8] in 1894 laid stress on this syndrome by means of case reports and pathologic studies. Important papers also were contributed by Goulden [9] in 1914 and by Beck [10] in 1927. Because of Devic's important contribution, this syndrome has been referred to as Devic's disease; it has also been called neuromyelitis optica or neuro-optica myelitis.

It is with a certain hesitancy that the following case report is offered. However, it is hoped that it may prove interesting not only by itself but as a basis from which several of the problems of this disease may be discussed.

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4. Hassin, G. B.: Histo-Pathology of the Peripheral and Central Nervous Systems, Philadelphia, W. B. Saunders Company, 1934.

5. Rivers, T. M.: Relation of Filtrable Viruses to Diseases of the Nervous System, Arch. Neurol. & Psychiat. **28**:757 (Oct.) 1932.

6. Albutt, T. C.: On the Ophthalmoscopic Signs of Spinal Disease, Lancet **1**: 76, 1870.

7. Erb, W.: Ueber das Zusammenvorkommen von Neuritis optica und Myelitis subacuta, Arch. f. Psychiat. **10**:146, 1879.

8. Devic, M. E.: Myélite aiguë dorso-lombaire avec névrite optique, Cong. franç. de méd. **1**:434, 1894.

9. Goulden, C.: Optic Neuritis and Myelitis, Tr. Ophth. Soc. U. Kingdom **34**:229, 1914.

10. Beck, G. M.: A Case of Diffuse Myelitis Associated with Optic Neuritis, Brain **50**:687, 1927.

REPORT OF A CASE

L. C., an intelligent and previously healthy Italian girl of 17, was admitted to the Lakeside Hospital, to the service of Dr. J. T. Wearn, on March 12, 1937. She was referred because of intense pains in the lower part of the back and legs, of five days' duration, and of increasing dimness in vision. Three weeks prior to her admission she had contracted a "cold" and had symptoms of coryza. This was followed by a backache, malaise and dizziness. One week later she had a temperature of 103 F. accompanied by severe pains radiating down the legs. Three days before entering the hospital she was troubled by pain in the right eye and gradual loss of vision. In the succeeding days she experienced difficulty in voiding.

The initial examination revealed a tall girl of 17 in a good state of nutrition, lying restlessly, with a temperature of 100.4 F., a pulse rate of 100 and a respiratory rate of 20. There was no evidence of infection of the respiratory tract, and the significant features were restricted to the neurologic study. The right eye was completely blind and the vision in the left eye appreciably diminished, particularly for colors. There were occasional spasmodic contractions which produced odd movements of the eyeballs; there was slight weakness of convergence. The right pupil was dilated and failed to respond to light, but the reactions of the left pupil were normal.

Examination of the right fundus revealed slight elevation of the nerve head, with blurring of the margins, and exudate and hemorrhages on its surface. The veins were engorged and tortuous, but the arteries were of average caliber. The left optic disk showed only slight haziness of its borders and moderate fulness of the veins. All four extremities were weak, and the tendon reflexes were hyperactive. The neck was slightly rigid, and the Kernig sign was positive. Light touch revealed hyperesthesia over the lower extremities. There was urinary retention. The laboratory findings as regards the spinal fluid were pathologic. The initial pressure was 250 mm. of water. The fluid was clear; the Pandy reaction, 2 plus; the lymphocyte count, 26; the protein content, 138 mg. per hundred cubic centimeters; the sugar content, 69 mg., and the colloidal gold curve, 2210.

The urine was normal, the white cell count being 11,400, with 74 per cent polymorphonuclears, 16 per cent lymphocytes and 8 per cent monocytes. Cultures of the blood were negative; cultures of the spinal fluid and inoculation of laboratory animals by Dr. Parker did not reveal a causative organism or virus.

The clinical course was at first progressively downhill. Within several days the vision in the left eye became more dim, until the patient was totally blind in both eyes. Both optic disks were elevated, about 3 diopters in each eye. The left nerve head was definitely blurred, with multiple hemorrhages and exudate.

Spinal fluid pressure at this time was 270 mm. of water. The Pandy reaction was 4 plus; the protein content, 191 mg. per hundred cubic centimeters, and the cell count, 75 lymphocytes.

At about this time there were increasing pains in the legs, with almost complete paralysis on the right side and partial paralysis on the left. Mentally, the patient became irritable, sometimes confused.

The sensory tests revealed patchy areas of diminished sensation, particularly in the lower extremities. Later, there was involvement of the arms, with sensory diminution and motor weakness. The abdominal reflexes were absent.

After the height of the illness was reached, about March 25, there was gradual improvement. On March 28 visual perception returned in the left eye, and several days later the patient could detect light in the right eye. In the subsequent days the vision improved steadily, and the pathologic changes in the eyeballs gradually

disappeared. On April 11 the patient could move her right leg, and on April 17 the reflexes had returned. On April 21 the patient was able to void voluntarily.

The patient was discharged on June 1, after three and a half months of illness, considerably improved. The vision in the right eye had returned to 6/12 and was 6/6 in the left eye. Studies of the visual fields showed moderate constriction for color and form in the right eye, while the left eye was normal. The optic nerve head on the right side showed moderate pallor, while the appearance of the left disk was essentially normal. The return of motor function was practically complete. The sensory losses had likewise cleared up, save for a residual area of hyperesthesia over the area of the lateral cutaneous nerve of the right leg. The personality disturbances had cleared up, and the patient appeared to be friendly and well adjusted.

During the two year period which has followed hospitalization, we have had occasion to make several subsequent examinations. The vision has remained normal in the left eye and only slightly impaired in the right. The fields are now complete, both for form and for color. Only slight pallor of the right disk remains. The paresthesia of the right side is another mild residual symptom.

Summary.—The patient was a girl in the late teens, who suffered from acute bilateral optic neuritis plus diffuse myelitis. The blindness preceded the definite signs of involvement of the spinal cord, which was manifested by complete paralysis of both legs, loss of sphincter control and marked hypesthesia of the legs and thighs. In the course of several months, the patient regained completely the vision in the left eye and had only a minimal impairment in the right. There was an almost total return of both sensory and motor function.

COMMENT

Differential Diagnosis.—In the case reported an early diagnosis of disseminated involvement, before the changes in the spinal cord appeared, would have been difficult. Cases of marked bilateral optic neuritis present a serious problem in the differentiation of these changes due to inflammation of the nerve and those resulting from the increased intracranial pressure associated with tumor of the brain. Goulden distinguished between those cases in which the involvement was strictly retrobulbar, with the alteration in the appearance of the disk, and those in which a true papillitis was present, due to changes in the most distal segment of the nerve. The ophthalmoscopic picture of such a papillitis may be of varying degree, from simple hyperemia to marked swelling with several diopters' elevation.

This difficulty in diagnosis was emphasized by Schaltenbrand,[11] who stated that bilateral optic neuritis may frequently be mistaken for evi-

11. Schaltenbrand, G.: Hirngeschwulstähnliche Erkrankungen die reine Geschwülste sind, Ztschr. f. d. ges. Neurol. u. Psychiat. **161**:162, 1938; abstracted, Year Book of Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1938.

dence of tumor of the brain. Walsh [12] called attention to 2 of 4 cases seen at the Johns Hopkins Hospital in which an erroneous diagnosis of tumor of the brain was made. Likewise, McKee and McNaughton [13] reported cases of papillitis, in 1 of which exploratory craniotomy was done and in the other of which ventriculographic examination was made.

In cases of optic neuritis the extent to which the edema spreads into the surrounding retina may be of diagnostic aid, but there are many instances in which the criteria suggested by Leslie Paton [14] may offer valuable assistance in excluding a lesion due to increased intracranial pressure. Paton stressed the fact that the loss of vision in optic neuritis precedes the ophthalmoscopic changes. The inference from this statement is that in the presence of edema of the optic nerve from papillitis blindness may be present, whereas ordinary papilledema precedes significant visual changes. This author added that in neuritis the changes in the fundi are of relatively short duration, the disks may show no trace of change after the swelling has subsided and the recovery of vision parallels the subsidence of swelling.

As to the danger of watchful waiting over a short period of time, to determine whether a questionable case of "choked disks" is due to optic neuritis, Gordon Holmes [15] observed that papilledema is too frequently considered an immediate danger to vision. Holmes stated: ". . . in even the most experienced hands operation, whether radical or palliative, entails considerable risk of life, and for this reason as well as on other grounds, as the lack of symptoms that can establish a definite localization, delay is often advisable." In our case the problem of differential diagnosis was rendered more difficult by the moderate elevation in the spinal fluid pressure, 250 mm. of water.

Treatment.—Obviously, because of the unknown causation, there is no specific treatment available, and many measures have been tried, with little or no success (Fralick and DeJong [16]). In this instance we found that large doses of salicylates were effective in alleviating the deep-seated pain in the back and legs. We believe exhausting treatment, such as fever therapy, to be contraindicated. The serious complication of paralysis of the bladder was treated by drainage and

12. Walsh, F. B.: Neuromyelitis Optica, Bull. Johns Hopkins Hosp. **56**:183, 1935.

13. McKee, S. H., and McNaughton, F. L.: Neuromyelitis Optica, Am. J. Ophth. **21**:130, 1938. Woods, A. C., in discussion on McKee and McNaughton, pp. 136-137.

14. Paton, L.: Papilledema and Optic Neuritis, Arch. Ophth. **15**:1 (Jan.) 1936.

15. Holmes, G.: The Prognosis in Papilledema, Tr. Ophth. Soc. U. Kingdom **57**:3, 1937.

16. Fralick, F. B., and DeJong, R. N.: Neuromyelitis Optica, Am. J. Ophth. **20**:1119, 1937.

irrigation with a mild antiseptic. The method introduced by Monroe and Hahn [17] was used. Nursing care was of course of tremendous value in the prevention of trophic ulcers and stasis.

Prognosis.—Many authors (Goulden, Rea [18] and Fralick and DeJong) give a mortality rate of 50 per cent for neuromyelitis optica. Those persons who survive may either recover completely or manifest a residual paralysis or some degree of secondary atrophy of the optic nerve. Fralick and DeJong observed that the rapidity of onset was not a deciding factor in the prognosis.

As regards the vision, the literature mentions the fact that "white disks" may occasionally be seen in the presence of perfectly normal vision. Whether this is due simply to atrophy of the nerve fibers or in how far it represents a proliferation of glial tissue or perhaps a diminution in the number of small blood vessels is not known. As King [19] has pointed out, Rönne would explain this retention of normal visual acuity in the presence of an apparent atrophy of the optic nerve as indicating the grossness of tests employing Snellen charts, which may permit registration of normal vision in the presence of the destruction of many of the nerve fibers.

Relation of Neuromyelitis Optica and Multiple Sclerosis.—Attention is directed particularly to retrobulbar neuritis, which is part of this syndrome. The incidence of unilateral retrobulbar neuritis as an early symptom of multiple sclerosis is well known. Adie [20] analyzed the figures for several London hospitals and concluded that in 38 per cent of cases this was the first symptom and in 58 per cent an early symptom. Lillie,[21] of the Mayo Clinic, studied some 500 cases and reported that disturbed vision was the initial episode in 15 per cent and a second or third episode in 35 to 40 per cent. The long duration of the natural remissions in multiple sclerosis makes the follow-up study in these cases sometimes extremely difficult. Adie reported that forty-seven years was the longest interval on record between the initial visual disturbance and the later onset of disseminated symptoms of multiple sclerosis. He therefore suggested that acute retrobulbar neuritis may, in some

17. Monroe, D., and Hahn, J.: Tidal Drainage of the Urinary Bladder, New England J. Med. **212**:229, 1935.

18. Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938, pp. 324-326.

19. King, C., in discussion on Kiely, C. E.: Neuromyelitis, Ohio State M. J. **32**:741, 1936.

20. Adie, W. J.: Acute Retrobulbar Neuritis in Disseminated Sclerosis, Tr. Ophth. Soc. U. Kingdom **50**:262, 1930.

21. Lillie, W. J.: Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17**:110, 1934.

instances, be the only manifestation of this disorder. Therefore, in the present state of knowledge the ultimate prognosis must be guarded.

We have reviewed the cases of encephalomyelitis seen at the University Hospitals during the ten year period from 1927 to 1937, so as to gain some perspective of the ultimate course in some of these cases. Of 5 cases reported by one of us (J. L. F.[22]) under the title of "Mild Migratory Myelitis," progressive symptoms characteristic of multiple sclerosis have developed in 3. Of 20 other cases in which the condition was considered encephalomyelitis, sequelae or later developments that could be considered multiple sclerosis have arisen in 5. Our opinion, therefore, concurs with that expressed by McAlpine,[23] who considered neuromyelitis optica to be but a syndrome of multiple sclerosis.

Although there is an apparent striking difference between neuromyelitis optica and chronic multiple sclerosis, the distinction may be merely that of the time element rather than an actual difference in etiologic agent or the pathologic process. In neuromyelitis optica the changes occur simultaneously or consecutively in a matter of several weeks, while in multiple sclerosis these same structures may be affected in the course of years. One may think of the analogy of this relation to various forms of tuberculosis; there may be galloping tuberculosis, with an acute pulmonic involvement, and chronic fibroid phthisis. In one instance the process may be acute and last a few weeks; in the other instance the disease process may extend over a period of many years, though the causation of the two conditions is the same.

CONCLUSION

We have presented briefly a case of neuromyelitis optica, which is interesting because of the marked changes in the vision and the eyegrounds, which subsequently disappeared almost entirely. The difficulties in distinguishing papillitis from papilledema are noted. We have used the designation of neuromyelitis optica with the understanding that this is but a descriptive designation for what is most likely but a form of multiple sclerosis.

22. Fetterman, J. L.: Mild Migratory Myelitis, Ohio State M. J. **32**:1090, 1936.

23. McAlpine, D.: Acute Disseminated Encephalomyelitis: Its Sequelae and Its Relationship to Disseminated Sclerosis, Lancet **1**:846, 1931.

VACCINIA WITH OCULAR INVOLVEMENT

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Many cases of vaccinia involving the lids and cornea have been reported, so that the reports of 2 additional cases of vaccinia of the lids, which are included in this article, are of comparatively little interest.

The purpose of this paper is to discuss measures that offer some promise of inhibiting the action of the vaccinia virus or that will protect the cornea against becoming involved.

The incidence of inoculation of the lids or cornea is apparently small when one considers the large number of vaccinations that are done each year. However, many cases are probably not reported, and possibly some may not be recognized.

When ocular involvement does occur, the lids are usually the first to become affected, and the cornea is probably inoculated later by the virus from the lesions on the lids or the conjunctiva. However, a number of cases have been reported in which there has been direct inoculation of the cornea.

It should be emphasized that because of an occasional careless inoculation of the eye after vaccination, one should not be influenced against vaccination. Evidently this excellent and simple method of preventing smallpox has not been taken advantage of as generally as it should be, for in 1938 the United States Public Health Service reported over 14,000 cases of smallpox in the United States. This should be of concern to the ophthalmologist when he recalls the serious impairment of vision suffered by many persons who were fortunate enough to recover from smallpox before the introduction of vaccination. No doubt there are still many eyes affected by this disease.

In 1919 Bedell [1] reported a case of vaccinia involving the lids and cornea, and his appended bibliography includes 92 cases with ocular involvement. In the same year Meder [2] reported a case in which the internal canthus and cornea were involved. In his bibliography there are 27 additional references.

1. Bedell, A. J.: Tr. Am. Ophth. Soc. **17**:273-281, 1919.
2. Meder, E.: Veröffentl. a. d. Geb. d. Med. **9**:405-434, 1919.

Ninety-one cases of vaccinia with ocular involvement were collected from the literature covering a period from 1919 to date.[3] In 32 of these the cornea was affected. Therefore, some impairment of vision probably occurs in approximately one third of the cases reported.

3. Amshel, F.: Vaccinia, Arch. Dermat. & Syph. **28**:287 (Aug.) 1933. Andervont, H. B., and Friedenwald, J. S.: Bull. Johns Hopkins Hosp. **42**:1-7, 1928. Anelli, D.: Boll. d'ocul. **11**:1265-1268, 1932. Aron: Klin. Monatsbl. f. Augenh. **74**:233, 1935. Ball, J. M., and Toomey, N.: Vaccinia of the Eyelids by Homo-Inoculation J. A. M. A. **79**:935-936 (Sept. 16) 1922. Benetazzo, G.: Dermosifilografo **11**:169-175, 1936. von Berger, F.: Ann. di ottal. e clin. ocul. **51**:262-266, 1923. Blessig, E.: Eesti Arst **11**:435-436, 1932; abstracted, Zentralbl. f. d. ges. Ophth **28**: 410-411, 1933. Busacca, A.: Lettura oftal. **2**:337-354, 1925. Camus, L., in discussion on Delord and Villard: Bull. Acad. de méd., Paris **97**:610-611, 1927. Colden: Klin. Monatsbl. f. Augenh. **74**:233, 1925. Cronstedt, L.: Upsala läkaref. förh. **31**:317-328, 1926; Lettura oftal. **8**:223-241, 1931. Espino, J. M.: Rev. cubana de oftal. **2**:253-256, 1920. Folk, M. L., and Taube, E. L.: Am. J. Ophth. **16**:36-39, 1933. Frei, W.: Zentralbl. f. Haut- u. Geschlechtskr. **39**:498, 1932. Friede, R.: Klin. Monatsbl. f. Augenh. **85**:427-430, 1930. Fuchs, A.: Wien. med. Wchnschr. **75**:2504-2506, 1925. Gabriélidès, A.: Des conjonctivites, Constantinople, Imprimerie "Estia," Galata, 1921. Gaté, J.; Michel, P.-J., and Boyer, C.-E.: Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **39**:46-48, 1932. Grüter, W.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:414-416, 1927. Hambresin: Bull. et mém. Soc. franç. d'opht. **41**:267, 1929. Horay, G.: Orvosi hetil. **67**:296-297, 1923; abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **11**:48, 1924. Jaensch: Klin. Monatsbl. f. Augenh. **74**:233, 1925. Jendralski, F.: ibid. **74**:233, 1935. Kerl, W.: Arch. f. Dermat. u. Syph. **148**:610-613, 1925. Lombardo, C.: Sperimentale, Arch. di biol. **75**:131-144, 1921. Ludwig, A.: Arch. f. Augenh. **109**: 346-352, 1935. Marín Amat, M.: Bull. et mém. Soc. franç. d'opht. **49**:85-90, 1936; Arch. de oftal. hispano-am. **32**:427-432, 1932; abstracted, Med. ibera **1**:73-74, 1932. Meyers: Ztschr. f. Med. beamte **35**:494-497, 1922. Montanelli, G.: Lettura oftal. **8**:135-142, 145-150, 153-158 and 161-165, 1931. Morax, V.: Précis d'ophtalmologie, ed. 4, Paris, Masson & Cie, 1931, pp. 215-217. Motolese, F.: Lettura oftal. **7**:336-338 and 341-343, 1930. Munns, G. F.: Am. J. Ophth. **14**:1037-1039, 1931. Nunn, L. L.: Northwest Med. **30**:194, 1931. Onfray, R.: Bull. et mém. Soc. franç. d'opht. **42**:267-268, 1929. Ortmann, K. K.: Hospitalstid. **75**:285-296, 1932. Pacelli: Not. di diagnost. e terap., 1929, no. 6. Portoghese, G.: Ann. di ottal. e clin. ocul. **62**:308-320, 1934. Sanguinetti, C.: Gior. di ocul. **3**:157, 1922. Schmelzer, H.: Ztschr. f. Augenh. **80**:356-359, 1933. Sédan, J.: Ann. d'ocul. **159**: 604-610, 1922. Sexe: Bull. et mém. Soc. franç. d'opht. **42**:262-269, 1929. Sezer, F. N.: Accidental Vaccination of Conjunctiva, Arch. Ophth. **20**:89-91 (July) 1938. Simidu, S.: Acta Soc. ophth. japon. **41**:167-172, 1937. Sinaiko, A. A.: Keratitis Postvaccinalosa, Arch. Ophth. **5**:91-92 (Jan.) 1931. Stark, A. M.: Arch. f. Dermat. u. Syph. **170**:38-60, 1934. Tessier, G.: Lettura oftal. **6**:137-142, 1929. Thies: Klin. Monatsbl. f. Augenh. **74**:227-228, 1925. Toomey, N.: Am. J. Ophth. **5**:292, 1922. Venturi, T.: Dermosifilografo **3**:576-582, 585-588, 591-594 and 597-601, 1928. Vita, A.: Atti d. cong. d'oftal. (1925), 1926, pp. 279-280. Wirtz, K.: Thesis, Cologne, no. 3, 1928, pp. 13-14 and 21-22. Young, C. A.: Virginia M. Monthly **52**:171-172, 1925.

REPORT OF CASES

The vaccinia of the lids in the following 2 cases occurred in children after the usual vaccination for smallpox.

CASE 1.—D. L., a girl aged 5 years, had an ocular lesion which was diagnosed as a sty by her physician. The unusual appearance of the lesion prompted a more careful inquiry into the history, and the following information was obtained from the mother.

Eleven days before the lesion on the lid appeared, the child had been vaccinated. Four days later the usual papule developed. Two days later a small lesion developed on the right lower lid, which gradually became yellowish. The lids became very red and swollen, so that the child was unable to open her eye. When she was first seen five days after the appearance of the lesion on the lid, examination showed the following picture:

The lids were red and swollen, and there was an ulcer 8 mm. long on the margin of the lid near the outer canthus. The surface of the ulcer was covered with a yellowish, soft, moist coating. Just nasal to this ulcer was another smaller one covered with a grayish coating. On the upper lid, in a corresponding position, were three small ulcers of similar appearance. The lids were indurated in the vicinity of the ulcers. There was no fluctuation, and when the scabs were removed a little serous fluid exuded but no pus. A smear and culture showed no specific organism. There were marked tenderness and swelling of the preauricular and submaxillary glands and some conjunctival secretion. The cornea was not affected. In two weeks the ulcers had healed, leaving slightly depressed scars with the loss of a few cilia.

CASE 2.—J. P., a girl aged 3½ years, was vaccinated (on the right thigh) eight days prior to the first visit. Three days after the vaccination a small red spot was noticed on the margin of the left lower lid, which gradually increased in size. Four days later the mother said that it developed into a sty and that the next day a similar lesion appeared on the upper lid, opposite the first.

The history of vaccination was not obtained until the nature of the lesion had been observed.

The appearance of the ulcers and induration of the lids was as described in case 1, with swelling and tenderness of the preauricular and submaxillary glands. Several more small ulcers developed on either side of the original ulcers along the margins of the lids and two small ones developed on the skin of the lower lid. All were covered with a moist gray coating. Five days later, or ten days after the first lesion appeared, the ulcers had healed, leaving slightly depressed scars and loss of cilia. The cornea was not affected.

The treatment was symptomatic, consisting of frequent gentle irrigations with boric acid solution, the application to the lids of an ointment containing mercury bichloride and the use of ice compresses. Trauma was particularly avoided.

During the two or three days when the lids were greatly swollen and difficult to open, no force or lid retractors were used because of the danger of inoculating the cornea. Cuffs were used on the child's arms to prevent her bending the elbows, so that she could not rub the eye.

The pustules on the lids were not as tender or painful, as is usually the case with a hordeolum.

COMMENT

Fortunately, the pustule and resultant ulcer on the lids is not followed by as much scarring as after the usual vaccination. Possibly this is due to the fact that there is less virus present and that it is continually kept moist by the tears and conjunctival secretion. Also, some immunity may develop from the original vaccination.

In cases reported in which the cornea became involved, keratitis resembling keratitis disciformis developed. It was usually centrally located and often accompanied by serous iritis.

The clinical picture of vaccinia involving the lids is characteristic; so with a history of recent vaccination or of the patient coming in contact with the virus, the diagnosis can be made with reasonable certainty. However, in case of doubt the laboratory tests can be of assistance.

Tanaka [4] first observed that a suspension of vaccine lymph undergoes flocculation when brought in contact with immune serum. He also observed that fluid from a serous effusion of lesions of smallpox which is left in contact with a suspension of vaccine lymph for several days at 37 C. produced a coagulation phenomenon akin to the Pfeiffer-Gruber-Widal reaction. These observations have been confirmed by others.

For the diagnosis of vaccinia or variola, both the complement fixation and the agglutination test may be used. Gordon [5] recommended doing both at the same time. The result of the complement fixation test is obtained on the first day and may be checked by the agglutination test on the following day. Both reactions appear to be specific for vaccinia and variola.

In the report of Gordon [5] and that of Craigie and Tulloch,[6] a description of the detailed technic of the tests is given.

A simple test for vaccinia is to inoculate the ear of a white rabbit with the vaccinial material. On the second day a red palpable swelling occurs. Increased vascularization is noted readily on looking through the ear when it is held up to the light. On the fourth day the site of inoculation appears as a firm, yellowish, elevated area. It soon becomes umbilicated, dries up, with scab formation, and disappears by the twelfth day.

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4. Tanaka: Centralbl. f. Bakt. (Abt. 1) **32**:726, 1902.

5. Gordon, M. H.: Studies of the Viruses of Vaccinia and Variola, Medical Research Council, Special Report Series, no. 98, London, His Majesty's Stationery Office, 1925.

6. Craigie, J., and Tulloch, W. J.: Further Investigations on the Vaccinia-Variola Flocculation Reaction, Medical Research Council, Special Report Series, no. 156, London, His Majesty's Stationery Office, 1931.

The obvious objection to this test is that it requires more time than the complement fixation and agglutination tests. However, antivaccinial serum necessary for the last-mentioned tests may not be available, and at least eight days are necessary for its preparation.

Inoculation of the rabbit's cornea is not satisfactory. According to Defries and McKinnon,[7] a positive take on the cornea occurs in only about 50 per cent of the rabbits. Fortunately, the cornea seems resistant to the virus.

With the definite diagnosis of vaccinia of the lids, one's chief concern is to try to prevent corneal involvement.

In 1896 Sternberg[8] observed that the serum of an animal rendered immune to vaccinia possesses the capacity of destroying that virus when brought in contact with it in vitro. This observation has been confirmed by others, and it is also true in the human being. The virus neutralization and virus agglutination properties of antivaccinial serums suggest the local use of the serum to inactivate the action of the virus and prevent involvement of the cornea.

Béclère, Chambone and Ménard[9] first reported a passive immunity in the fetus from the mother, which has been confirmed by others. Sato[10] reported that young rabbits of vaccinated mother rabbits possessed corneal as well as cutaneous immunity.

Hlava and Honl[11] reported that the subcutaneous injection of the serum of immunized calves protected normal calves against subsequent inoculation with the vaccinia virus. Similar injections in school children of 0.6 to 1 cm. of immune serum per kilogram of body weight produced protection against subsequent inoculation with vaccinia. This has been confirmed by others.

Camus,[12] and Henseval and Convent[13] pointed out that the preventive action of immune serum was greater than the curative action. By multiple vaccinations, Gordon[5] was able to produce more immune serum, so that smaller doses could be used.

From these observations it seems logical to expect that some immunity would be produced by the injection of immune serum. If the serum could be injected as soon as the diagnosis of vaccinia of the lids is made, sufficient immunity might be produced to prevent corneal involvement.

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7. Defries, R. D., and McKinnon, N. E.: Am. J. Hyg. **8**:107, 1928.
8. Sternberg: Centralbl. f. Bakt. (Abt. 1) **19**:857, 1896.
9. Béclère, A.; Chambone, and Ménard: Ann. Inst. Pasteur **13**:81, 1899.
10. Sato, K.: Ann. Inst. Pasteur **35**:481, 1921.
11. Hlava, J., and Honl, I.: Wien. klin. Rundschau **9**:625, 643, 1895.
12. Camus L.: Compt. rend. Soc. de biol. **73**:294, 1912.
13. Henseval, M., and Convent, A.: Bull. Acad. roy. de méd. de Belgique **26**: 251, 1912.

We were unable to demonstrate this satisfactorily with rabbits, because the cornea is apparently resistant to the vaccinia virus. However, sufficient immunity may be produced in the rabbit to prevent a take on the ear, while with rabbits not previously immunized a good take is practically always obtained.

The virus of vaccinia is made inactive by several drugs. Gordon [5] found that potassium permanganate in dilutions up to 1:100,000 in contact with the virus for one hour at room temperature (17 C. [62.6 F.]) inactivated the virus. Also, mercuric chloride in a dilution up to 1:10,000 destroyed the virus.

Kligler and Bernkopf [14] reported that relatively small amounts of ascorbic acid inactivated many times the infective dose of the vaccinia virus. A dilution of 1:2,000 inactivated one hundred times the infective dose of the virus when incubated for three hours. They suggested that the ascorbic acid acts as an oxidation-reduction substance.

As previously stated, the aim of this study was to try to find some method of inhibiting the action of the virus and to prevent another take after the person or animal had been successfully inoculated. The ears of white rabbits were found to be the most satisfactory site for inoculation and observation, so a series of rabbits were used with controls in each group.

Sulfanilamide and neoprontosil (disodium 4-sulfamidophenyl-2′-azo-7′- acetylamino-1′-hydroxylnaphthalene-3′, 6′ disulfonate) were the first drugs used. The rabbits were inoculated with the vaccinia virus. At the first appearance of satisfactory inoculation, usually the second to the fourth day, the drugs were given. In all the animals the pustules followed the same course and size as in the controls. To another series of rabbits the drugs were first administered for several days until they became cyanosed, indicating a high concentration of the drugs. Then they were inoculated with vaccinia virus, and in all a satisfactory take occurred. Therefore, we concluded that the action of the virus was not inhibited by sulfanilamide or neoprontosil. Sulfapyridine was used in the same way, but it did not inhibit the action of the virus.

A number of drugs, such as tincture of iodine, metaphen and silver nitrate, were used as follows: After the epidermis was abraded, the drug was applied to the site and then the virus. No takes occurred when the tincture of iodine or metaphen was used. In another series the ears were vaccinated in the usual way, and wet dressings with ascorbic acid were applied. In these, no take occurred.

Recently Friedenwald [15] reported that ascorbic acid is normally found in both the blood and the aqueous and that the concentration in the

14. Kligler, I. J., and Bernkopf, H.: Nature, London **139**:965-966, 1937.
15. Friedenwald, J. S.: Tr. Am. Ophth. Soc., 1939, to be published.

aqueous is ten times greater than it is in the blood. Since ascorbic acid has been found to inhibit the action of the vaccinia virus, administration of ascorbic acid to increase the saturation in the blood and the aqueous might possibly offer some added protection to the cornea.

SUMMARY

No cases of vaccinia with ocular involvement have been seen since this study was started. Therefore, no definite conclusion as to the value of these measures in the prevention of corneal involvement in the human being has been made. However, they suggest a method of treatment that might offer some promise of preventing corneal involvement. It is most distressing to watch the progress in such cases, while treating the lesion symptomatically and hopefully praying that the cornea will not become involved.

In the treatment of vaccinia with ocular involvement it would seem rational to use *some of the following measures:* First, one should avoid trauma that might cause inoculation of the cornea, and great care should be exercised in opening the swollen lids. For young children, cuffs should be used to prevent them from bending the elbows and thus rubbing the eyes with the fingers. Such cuffs may also be used to prevent them from touching or scratching the original vaccination site, and thus carrying the virus to the eyes.

As soon as the lesion on the lids is diagnosed, potassium permanganate, tincture of iodine, metaphen or mercury bichloride should be applied to the lesion.

Ascorbic acid may be administered generally and locally.

The patient may be inoculated again with the vaccinia virus to increase the immunity or virulicidal substance.

Antivaccinal serum may be applied locally to inactivate the virus present on the margins of the lid and in the conjunctival secretion. The immunity may also be increased by the injection of immune serum.

EXPERIMENTAL RETINOBLASTOMA

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The present investigation was suggested by previous experimental work on the effect of carcinogenic substances on neural structures.[1] It was thought that the retina, as an easily accessible neural organ, should lend itself better to such study than the brain itself.

The first attempt to study the effect of carcinogenic substances on the eyeball was made by Schreiber and Wengler.[2] On the basis of the discovery of B. Fischer, who had demonstrated the carcinogenic properties of scarlet red, they injected a concentrated solution of this dye in olive oil into the anterior and posterior chambers of the eyes of rabbits and dogs. They described the new formation of large ganglion cells in the retinas of these animals about one hundred and twenty days after the injection and concluded that adult cells of the retina are able to divide mitotically. Unfortunately, no actual photomicrographs were added to their publication. It is surprising that despite the progress made in the experimental study of tumors in general no reports could be found in the literature of the last decade dealing with the application of these new discoveries on the eye.

In the present investigation two carcinogenic substances were originally used, 1,2,5,6-dibenzanthracene in lard solution and styryl 430 (2[p-aminostyryl] 6 [p-acetylaminobenzoylamino] quinoline methoacetate) in aqueous solution The latter did not give any results. It soon became evident that in the control rats lard alone produced intense proliferation of the different tissues, corroborating the carcinogenic action of lard, olive oil and other fatty mediums in other organs as

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Read as a preliminary paper at a meeting of the Chicago Ophthalmological Society, May 8, 1939.

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1. Weil, A.: Experimental Production of Tumors in the Brains of White Rats, Arch. Path. **26**:777 (Oct.) 1938.

2. Schreiber, L., and Wengler, F.: Ueber Wirkungen des Scharlachöls auf das Auge speziell auf die Netzhaut, Mitosenbildung der Ganglienzellen, Arch. f Ophth. **74**:1, 1910.

reported by Burrows, Hieger and Kennaway.[3] Consequently, the attempt was made to fractionate lard into different compounds by fractionate distillation in vacuo (up to 250 C.) and by saponification and to test the action of the different substances on the eye of the rat. Other substances which were tested were olive oil and iso-oleic acid (supplied by C. J. Farmer).

Altogether, until the date of submission of this paper 34 rat eyes have been studied, the rats having a maximal survival period of eleven months. The present paper is concerned mainly with experiments of injecting lard and olive oil into the corpus vitreum of the rat eye.

TECHNIC

The lard (Swift's) was heated in vacuo (water suction pump) to a temperature of 250 C., and the distillate was separated from the residue. The different fractions of the distillate did not prove to be active, and, therefore, in the final experiments the residue only was used. The material to be tested was sterilized for one hour at 120 C. and then cooled to about 40 C. White rats weighing between 150 and 180 Gm. were used. After the induction of light ether anesthesia, the injection was made through the sclera and the retina into the corpus vitreum, a long needle (no. 27) being used. The number of drops injected could easily be controlled. The rats recovered soon, and except for a mild transitory conjunctivitis no external lesions remained. The transparent fat soon changed into a yellowish granular mass, and the eye became atrophic. The rats were killed at different periods; the eyeballs were fixed in solution of formaldehyde U. S. P. or in Bouin's solution. They were embedded in paraffin, cut in a sagittal plane at 10 microns in serial sections and stained with different methods.

RESULTS

One week after the injection of superheated lard a cellular granulation tissue appeared which filled the corpus vitreum. At the points of contact with the retina the cells of the ganglion cell layer had increased considerably in number. In sections impregnated with a silver stain the majority of these newly formed cells showed a large vesicular nucleus, surrounded by argyrophilic cytoplasm (fig. 4 *B*). In preparations stained with cresyl violet the nucleus contained a fine, dustlike chromatin and a tiny nucleolus. While normally only one layer of cells is present with nuclei measuring from 6 to 9 microns, after injections of superheated lard there appeared several layers of large cells with nuclei measuring from 10 to 12 microns (fig. 4 *A*).

At the early stages after the injections of superheated lard, which were studied at eight, fourteen and twenty-nine days, respectively, the cytoarchitecture of the retina was well preserved, and there was only

3. Burrows, H.; Hieger, I., and Kennaway, E. L.: Studies in Carcinogenesis, J. Path. & Bact. **43**:419, 1936.

Fig. 1.—Sagittal section through a rat eye into which superheated lard had been injected. Van Gieson's stain. *A*, appearance one hundred and twenty-nine days after the injection. Magnification, × 24. The retina is hypertrophic, and the corpus vitreum is replaced by granulation tissue. The lens has been removed. *B*, appearance one hundred and fifty-four days after the injection. Magnification, × 26. There is marked hypertrophy of the retina with loss of differentiation between the inner and the outer nuclear layer. The granulation tissue contains large multinuclear cells surrounding acicular spaces.

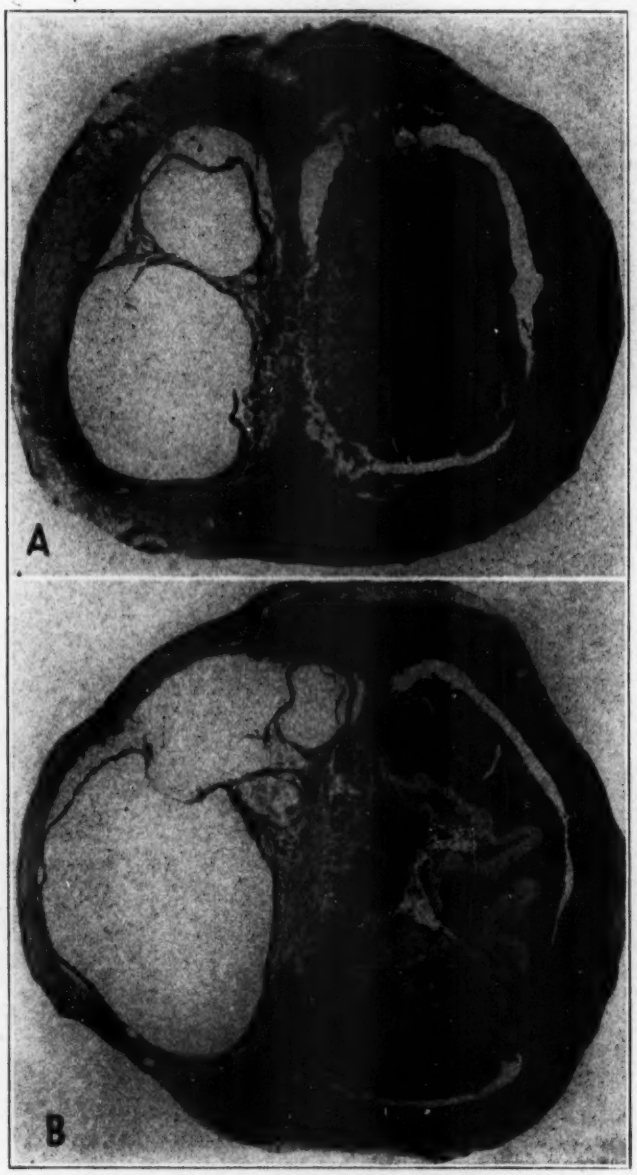

Fig. 2.—Sagittal section through a rat eye into which superheated lard had been injected two hundred and eighteen days previously. Van Gieson's stain; magnification, × 23. The lens had been removed. *A*, section through the outer third of the eyeball. *B*, section through the center. There is a considerable proliferation of the retinal cells which fills out the space between the sclera and the lens. The former granulation tissue has been transformed into an atrophic connective tissue scar. The outlines between the inner and the outer nuclear layer have disappeared, and cell masses are seen invading the inner reticular layer.

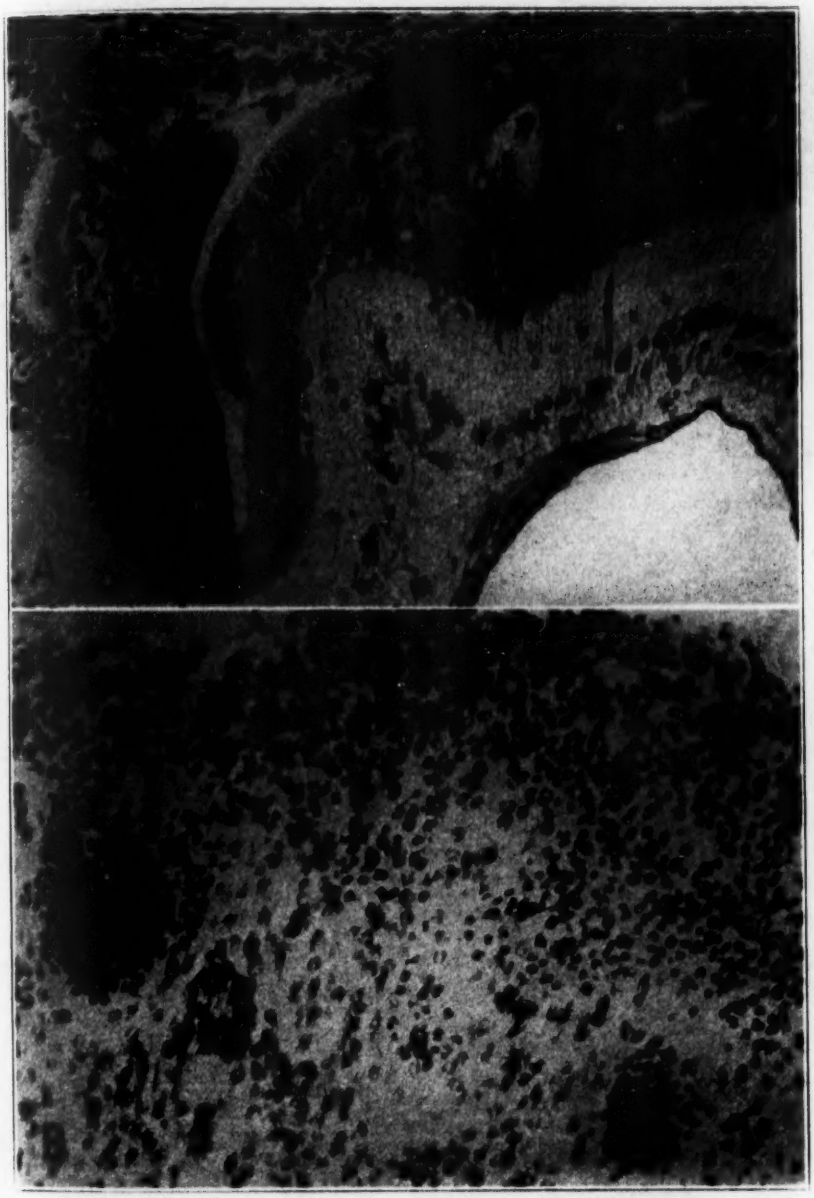

Fig. 3.—Section through the retina of a rat eye into which olive oil had been injected one hundred and eighty-nine days previously. Van Gieson's stain. In *A*, the hypertrophic retina is folded, and long tubes are formed, lined at the inner surface by rods and cones. Transverse sections of these tubes give the impression of rosettes, two of which are seen in the right upper corner. They are surrounded by a cluster of small nuclei and contain large scavenger cells. Magnification, × 300. In *B*, germinal centers appear in the inner nuclear layer with great numbers of mitotic figures. Newly formed cells are seen migrating from the outer into the inner nuclear layer. Magnification, × 375.

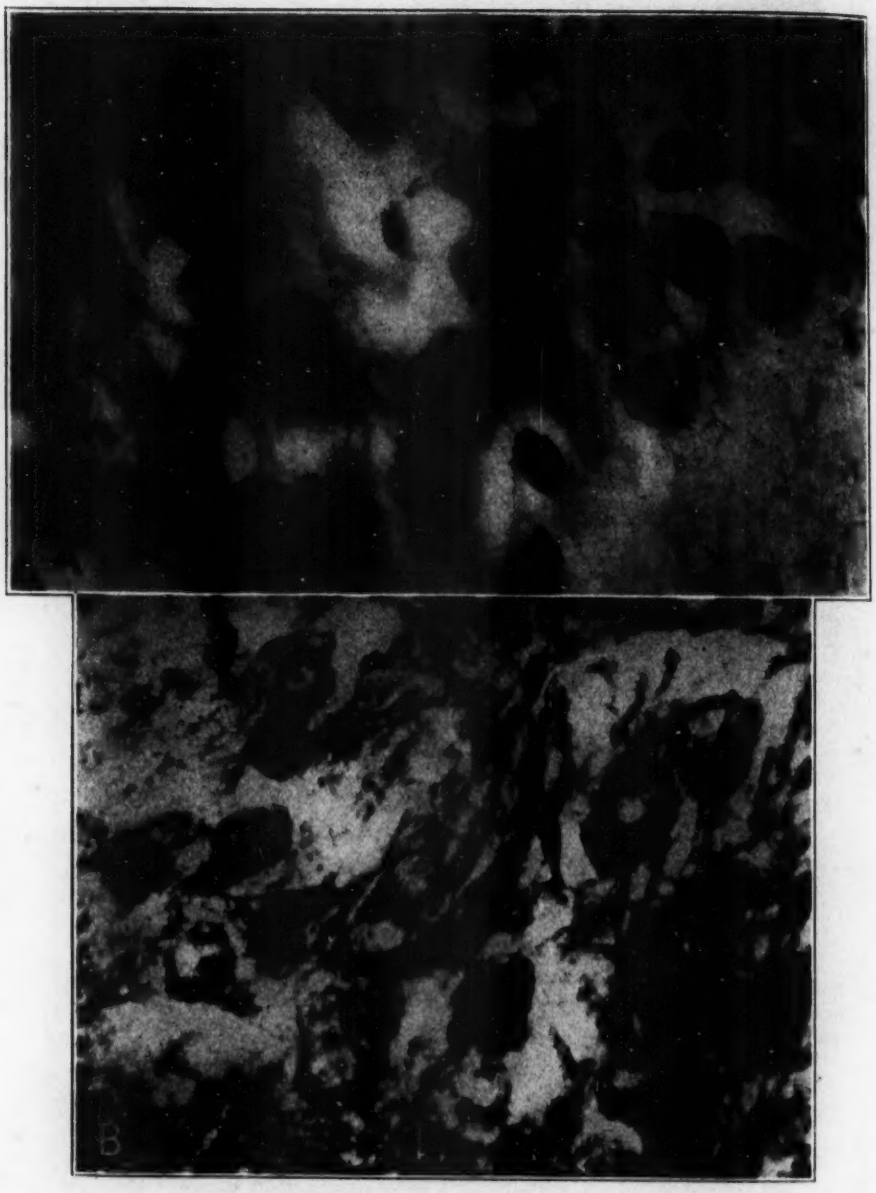

Fig. 4.—Proliferated cells in the ganglion cell layer. *A*, a rat eye into which iso-oleic acid had been injected one hundred and thirteen days previously. Cresyl violet stain; magnification, × 1,200. The large nuclei contain a fine granular chromatin and a nucleolus. The cytoplasm stains well but does not show a definite structure. *B*, a rat eye into which olive oil had been injected one hundred and eighty-nine days previously. Davenport's stain; magnification, × 1,490. The vesicular nuclei are surrounded by an argyrophilic cytoplasm.

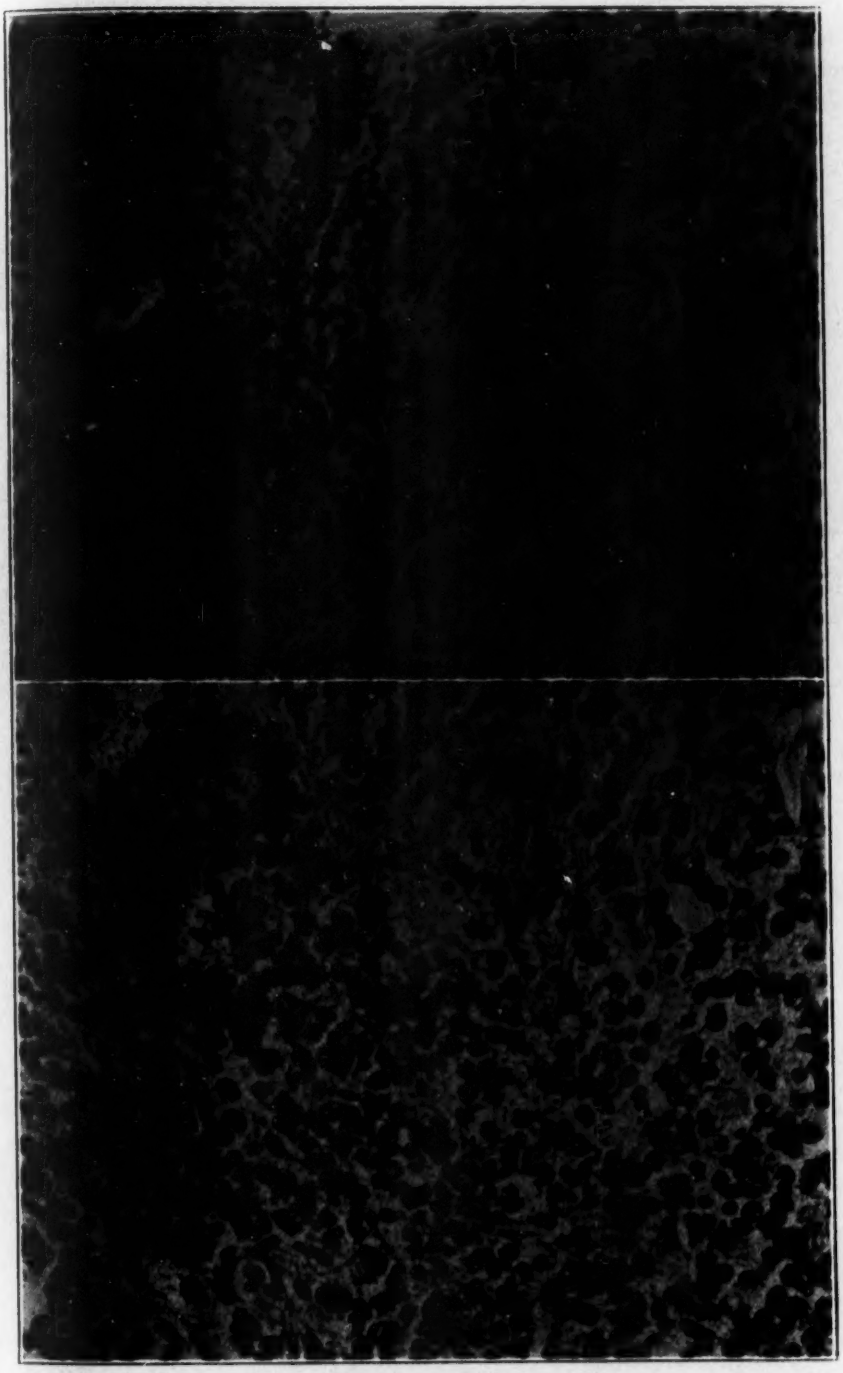

Fig. 5.—Retina of a rat eye into which superheated lard had been injected two hundred and eighteen days previously (same eye as shown in fig. 2). Van Gieson's stain; magnification of *A*, × 250; of *B*, × 480. The newly formed cells are seen invading both nuclear layers and the inner reticular layer. They have a tendency to cluster around blood vessels or to form circles around structureless tissue.

a mild edema at the points of contact with the granulation tissue. In the latter there appeared around the third week giant multinuclear cells, rich in cytoplasm, which surrounded acicular spaces, like those described in brains of rats into which lard was injected.[1] After three months the rods and cones and the outer and inner nuclear layers were well

Fig. 6.—Same eye as in figures 2 and 5. Cresyl violet stain; magnification, × 1,720. Numerous mitotic figures are seen at different stages.

preserved and well separated. There was, however, a considerable increase in the size of the retina as a whole to two to three times its original size, of the inner reticular layer and of the number of cellular elements. At this stage ten and more rows of nuclei could be counted

in the inner nuclear layer, as compared with a normal figure of five rows. In addition, the size of the nuclei of the inner nuclear layer had increased from normally 5 or 6 microns to 8 or 9 microns (fig. 1 *A*).

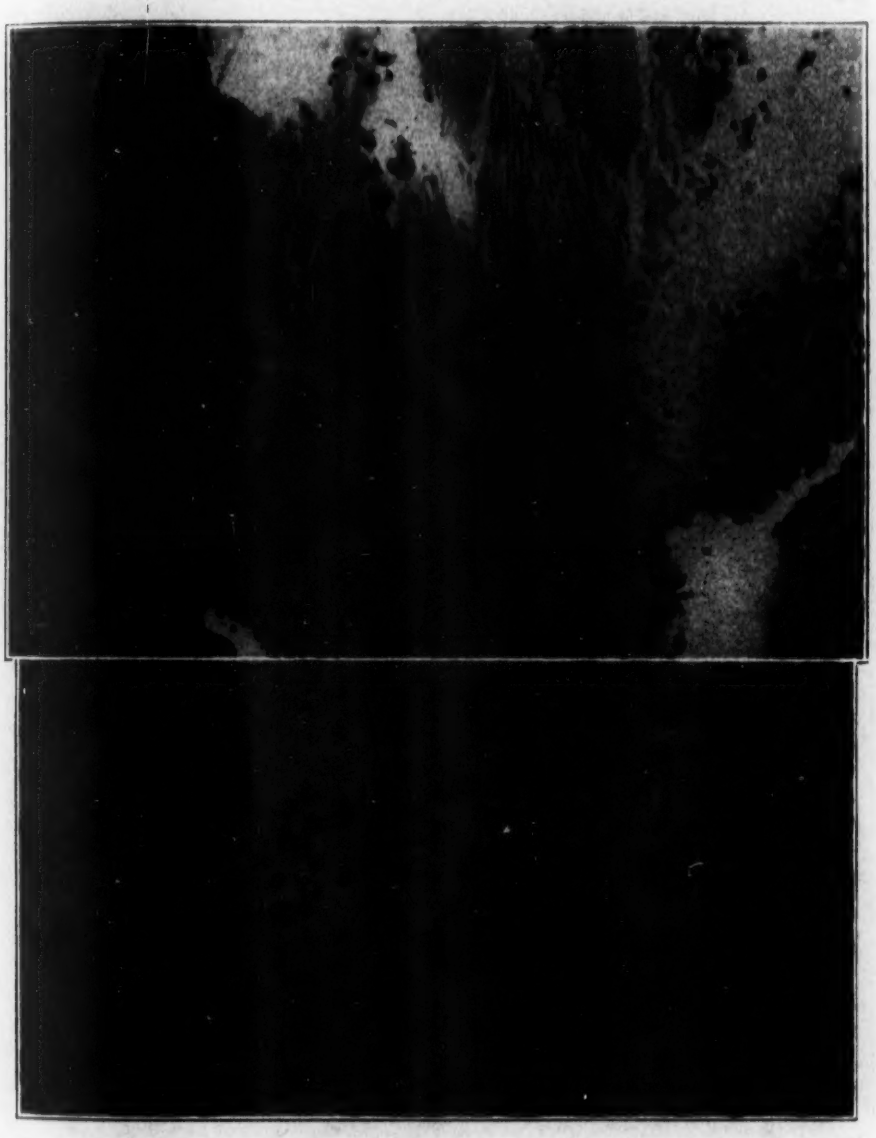

Fig. 7.—Same eye as in figures 2, 5 and 6. Van Gieson's stain. *A*, newly formed retinal cells are seen invading the optic nerve. Magnification, × 203. *B*, newly formed cells are seen invading the inner reticular layer. Magnification, × 328.

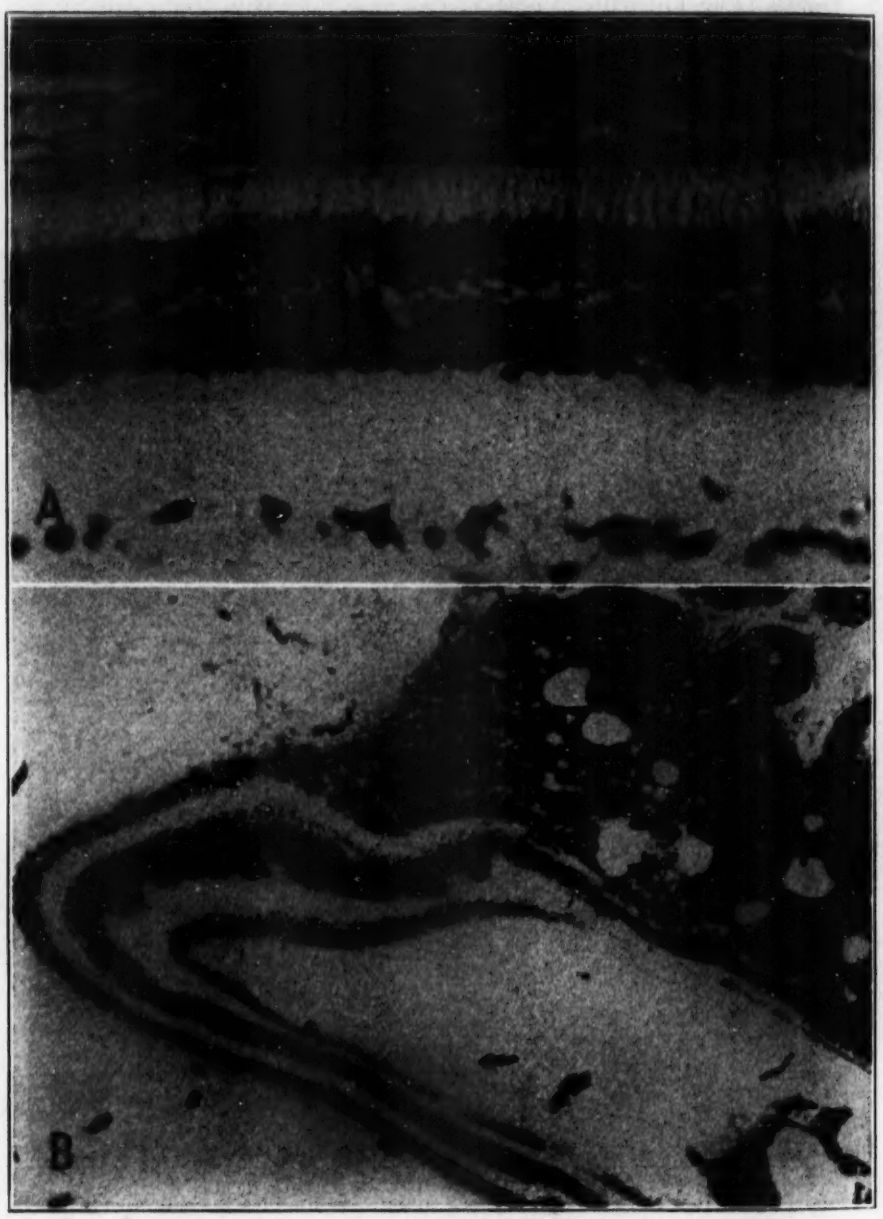

Fig. 8.—*A*, section through the normal retina of a rat eye. Van Gieson's stain; magnification, × 485. *B*, section through a human retinoblastoma. Hematoxylin-eosin stain; magnification, × 44. Part of the retina has retained its normal appearance. The tumor seems to originate from the nuclear layer.

Frequently rosettes were found surrounded by the tiny cells of the outer nuclear layer. Their formation is easily explained if one studies figure 3. The increase in the size of the retina necessarily leads to a process of folding and tubule-like formations, lined inside by a layer of rods and cones. The latter gradually disintegrated, and their debris was taken up by large scavenger cells, which may be seen in the center of the two rosettes at the right of the illustration. The smaller cells surrounding the rosettes proliferated, and around the fifth month after the injection numerous mitotic figures appeared (fig. 6). The newly formed cells were seen migrating into the inner nuclear layer, and the

Fig. 9.—Same eye as in figures 3 and 4 *B*. Davenport's stain; magnification, × 630. A typical rosette is seen with a large phagocytic cell in the center, surrounded by rods and cones.

separation between the two layers gradually disappeared. About the sixth month after the injection there began a migration of cells into the inner reticular layer (fig. 7 *B*), and finally the whole retina was transformed into a cellular structure, which expanded into the posterior chamber after the shrinkage of the original granulation tissue and the connective tissue scar which replaced it (fig. 2). In this tumor formation there was in places a tendency of cells to cluster around blood vessels or to form circles around a structureless area (fig. 5 *B*).

The papilla of the optic nerve showed swelling in the early stages. During the seventh month proliferated retinal cells were seen invading the optic nerve (fig. 7 *A*). The ciliary body showed marked hypertrophy

at about the fourth month after the injection; both papillae as a whole as well as the covering epithelium were enlarged. In 1 case a remarkable proliferation of the epithelium was observed after injection of superheated lard with the formation of detached long rows of epithelial cells. The iris showed early cellular infiltration, which persisted during the later stages. The lens in some cases underwent early degeneration. Its capsule appeared thickened, and the interior was filled with a dense accumulation of phagocytic cells. In other cases it merely was shrunken, and its structure was well preserved (fig. 1). The choroidea of the early stages was shrunken, though the pigmented cells were preserved.

Fig. 10.—Same eye as in figure 1 *B*. Van Gieson's stain; magnification, × 230. Hyperplasia of the ciliary body is evident.

Five months after the injection its blood vessels became wider, but there was apparently no proliferation of the cells of the pigmented cell layer. The sclera and cornea were not changed to any marked degree, except for a mild thickening observed during the later stages.

The results following injections of olive oil and iso-oleic acid were not as uniform as those following injections of superheated lard. Iso-oleic acid produced an early inflammatory reaction, which in 1 case destroyed the retina. In another case in which olive oil was injected, no proliferative changes were found seven months after the injection, though the possibility had to be considered that most of the oil escaped

after the injection. In 1 case active proliferation and mitosis of retinal cells was observed one hundred and eighty-nine days after the injection of olive oil.

COMMENT

If one compares the present experiments with the early attempts of Schreiber and Wengler to produce tumors by injecting scarlet red in olive oil, their failure may be explained in two ways. First, they made the injections into the anterior chamber in most of their experiments and there was no direct contact between the carcinogenic substance and the retina. Second, they did not extend their experiments long enough but interrupted them at one hundred and twenty days. Judging from the present observations, there is no doubt that they actually observed the beginning of mitotic divisions; unfortunately, no actual photomicrographs were added to their paper. But the preceding descriptions demonstrate that active mitosis and neoplastic transformation begin to appear after the fifth or the sixth month only. This experience is in contrast to that obtained in experiments in which cutaneous tumors followed injections of dibenzanthracene, about four months apparently being sufficient to produce an active growth. Brain tissue seems to be still slower in responding to carcinogenic agents.

It is difficult to state which of the two nuclear layers of the retina showed the more active cellular proliferation. It seemed that the inner layer first increased in width, but that the outer layer participated with active mitosis only during about the sixth month. The experimental production of the "Wintersteiner rosettes" is not completely in agreement with the commonly held opinion that in the human eye they are formed during the neoplastic process and represent primitive neural tubes or a primitive optic vesicle. It was previously pointed out that they are formed early after the injection of superheated lard as invaginations of the enlarged retina. They imitate tubular formations, lined by rods and cones. The latter may persist within such a rosette for some time before they disintegrate. The large scavenger cells within the rosettes have been described as giant astrocytes or ganglion cells which send out fine processes. Sections impregnated with silver stain, however, demonstrate that these processes are fine, cilia-like extensions arising from the inner wall of the rosette. Furthermore, such preparations demonstrate clearly that two thin membranes separate the rods and cones from the smaller nuclei and the latter from the outer nuclear layer, respectively (fig. 9).

Attempts to impregnate the neoplastic cells with silver stains were not successful. Occasionally a tiny layer of argyrophilic cytoplasm could be seen around the vesicular nuclei or a short process of the cytoplasm. Impregnation of the proliferated cells of the ganglion cell

layer was more successful and demonstrated clearly newly formed neurons (fig. 4). No marked gliosis could be seen in preparations stained by the Holzer method, even in experiments of seven months' duration. There was also no increase in vascularization, and no necrotic areas had been formed up to this stage.

While in regard to the early experiments of up to five months' duration some doubt might exist whether one should classify the retinal proliferation as "granulomatous" or "traumatic reaction," there is no doubt that superheated lard induces a true neoplastic process six and more months after its injection into the corpus vitreum of the rat eye. The term retinoblastoma instead of neuroblastoma of the retina has been preferred for this tumor formation, in adherence to Verhoeff's terminology, in order to emphasize the primitive character of the proliferated cells.

SUMMARY

Injections of superheated lard and of olive oil into the corpus vitreum of the eyes of white rats led to neoplastic disease of the retina. The cells of the ganglion cell layer which were in direct contact with the injected substance showed active proliferation during the first month.

Both the inner and the outer nuclear layer responded with cellular proliferation, and active mitosis appeared in the latter about the sixth month after the injection. At the same time invasion of the optic nerve by proliferated retinal cells was observed.

The term retinoblastoma has been applied to this tumor formation in order to describe the primitive character of the proliferated cells.

GLIOMA OF THE OPTIC NERVE

A CRITICAL REVIEW; REPORT OF TWO CASES, WITH AUTOPSY OBSERVATIONS IN ONE

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While 1 or 2 cases of primary glioma of the optic nerve are reported each year in the literature, it is the clinical aspect that almost always predominates in these reports. Relatively little has been said regarding the pathologic study and classification of these tumors [1] since the introduction of the impregnation methods of Cajal and del Rio-Hortega. Save for Dandy's paper [2] in 1922, there has been no critical evaluation of the surgical procedures. It is, then, with the pathologic aspect and also with the operative approach that this paper is chiefly concerned.

In general, these tumors are divided into two large groups: (1) the dural endotheliomas or meningiomas, which arise from the fibrous sheath of the nerve, and (2) the gliomas, which arise from the neuroglial elements within the nerve itself.

Clinically, the meningiomas seem to be more malignant than the gliomas, a fact recognized by Hudson [3] in 1912. Twenty-nine cases were cited in his paper. Nineteen of the patients were followed for less than one year. Of the remaining 10, 2 died of meningitis at operation, 4 died of recurrences and 4 were followed for from one to six years without symptoms of recurrence.

Two of the cases in which there were recurrences are cited. In the case reported by Byers,[4] recurrence caused death nine years after

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1. del Rio-Hortega, P.: Estructura y sistematización de los gliomas y paragliomas, Arch. españ. de oncología **2**:569, 1932; Anatomía microscopica de los tumores del sistema nervioso central y periférico, Madrid, S. A. Blass, 1934, p. 154.

2. Dandy, W. E.: Prechiasmal Intracranial Tumors of Optic Nerves, Am. J. Ophth. **5**:169, 1922.

3. Hudson, A. C.: Primary Tumours of the Optic Nerve, Roy. London Ophth. Hosp. Rep. (pt. 3) **18**:317, 1912.

4. Byers, W. G. M.: Primary Intradural Tumors of the Optic Nerve: Fibromatosis Nervii Optici, in Studies from the Royal Victoria Hospital, Montreal, Toronto, J. A. Carveth & Co., 1901, no. 1, p. 3.

primary removal of the tumor; there was a recurrence in the orbit one year after the first operation, at which time exenteration was done. The patient, a girl, lived eight more years, and autopsy revealed extensive intracranial involvement.

The other case is reported by Pagenstecker.[5] A local recurrence appeared twenty-five years after the original operation, death from thrombosis of the cavernous sinus following the second removal. At autopsy it was found that the tumor had not advanced as far as the chiasma.

Of considerable interest, though not of importance to this paper, are the bilateral intracranial meningiomas involving the optic nerve described by Schott [6] and Dandy.[2]

Among the cases of glioma, we found nothing comparable to these cases of meningioma in which recurrence took place nine and twenty-five years later. The course seems rather to be benign. In Lundberg's case 1 [7] an oligodendrocytoma was thought to have been incompletely removed, and the patient was in good health seventeen years later. The patient in Byers' case 1 [4] was alive and well after fifteen years, although at operation the growth was found to extend up to the optic foramen. The patient in Seefelder's case 1 [8] showed no evidence of recurrence in ten and one-half years, nor did de Schweinitz' patient [9] in seven years. It is not known whether in the last 2 cases the growth was thought to be completely removed. Only in the case of Barraquer [10] was there intracranial extension after an appreciable lapse of time (nine years).

Such, then, was the general concept of the problem under which the following 2 cases were handled.

REPORT OF CASES

CASE 1.—*History.*—M. H., an 8 year old Negro girl, was admitted to the hospital on June 23, 1936, because of exophthalmos of the right eye of two years' duration. The past and family histories were unimportant. The exophthalmos

5. Pagenstecker, A. H.: Ueber Opticustumoren, Arch. f. Ophth. **54**:300, 1902.

6. Schott: On Some Affections of the Optic Nerve, Arch. Ophth. **6**:276, 1877.

7. Lundberg, A.: Le gliome primitif du nerf optique et du chiasma des nerfs optiques, Arch. d'opht. **1**:97, 1937.

8. Seefelder, R.: Beiträge zu den Gliomen des Sehnerven, Wien. klin. Wchnschr. **44**:838, 1931.

9. de Schweinitz, G. E.: A Contribution to the Subject of Tumors of the Eyelid and Orbit, Tr. Am. Ophth. Soc. **14**:341, 1915-1916.

10. Barraquer, J.: Mixoma quistico del nervio optico de la papila y retina derechas y de la cavidad craneal y órbita izquierda, Arch. de oftal. hispano-am. **2**: 132, 1902.

was gradual in onset and progressive. There had been no ocular pain or headache. Vision was noted to have been lost about two months before the patient came to the hospital. There had been no difficulty with the left eye. The patient's general health had been good.

Examination.—There was marked exophthalmos of the right eye. Vision was limited to perception of light. Motion was partially limited on internal rotation. The globe could be entirely displaced, but no distinct mass could be felt in the orbit. However, the retrobulbar space appeared to be filled with nonpulsating undifferentiated tissue. The lids, conjunctivas, sclera, cornea, iris and media were normal. The pupil was partially dilated but reacted consensually. The right optic disk showed primary atrophy.

Vision in the left eye was 20/20, and examination otherwise gave entirely negative results.

General physical and neurologic examinations gave negative results. The Wassermann reaction was negative, and the blood and urine were normal. Roentgenograms of the skull showed the right optic foramen to be dilated, measuring 10 mm., while the left measured but 4 mm. There was no evidence of bone erosion.

A diagnosis of retrobulbar tumor was made, and the patient was prepared for operation.

Operations.—Exploration of the orbit was done with the patient under ether anesthesia. A small hard tumor measuring 5 by 2 cm. was removed; it was entirely within the muscle cone and extended to the apex of the orbit, apparently encapsulating the optic nerve. Because of the type of tumor first reported, craniotomy for removal of the intracranial portion of the tumor was deemed necessary, and this was attempted by a member of the neurosurgical service three weeks after the first operation. A right anterior trephine opening was made, but when the dura was incised excessive bleeding was encountered. Further operative procedures were postponed until one week later, when a firm, circumscribed, pinkish tumor, 1.5 by 1 cm. in size, was found to protrude from the optic foramen posteriorly toward the optic chiasma. It was not attached except at the foramen. The optic nerve was not involved as far as the chiasma but seemed to fray out into the tumor and was easily divided with the foramen hook. The tumor was readily removed with a sharp curet. It was felt that some of the tumor remained in the optic foramen, and because of inadequate exposure attempts were made to remove it with a curet. Considerable arterial bleeding resulted from this procedure, and it was necessary to insert a pack to control it. After the bleeding was controlled, another attempt was made to remove the tumor from the optic foramen, but bleeding again occurred, and the operation was terminated after the hemorrhage was controlled with packs. The wound was closed, and the patient returned to the ward in poor condition. She failed to respond after the operation and died six hours later. Autopsy was done, and the results are reported as follows:

Pathologic Study.—The first surgical specimen was a soft, conical, encapsulated tumor mass, measuring 5 cm. in length and varying from 0.6 to 2 cm. in diameter. The cut surface was uniformly grayish white. In the region of the apex of the conical mass the optic nerve was seen, surrounded by a collar of neoplastic tissue (fig. 1 *A*). As further sections were made, the nerve gradually became lost in the mass at the base of the tumor.

The second surgical specimen consisted of several fragments of soft grayish white tissue, the largest of which measured about 1 cm. in diameter. Both specimens were fixed in a 10 per cent dilution of solution of formaldehyde U. S. P.

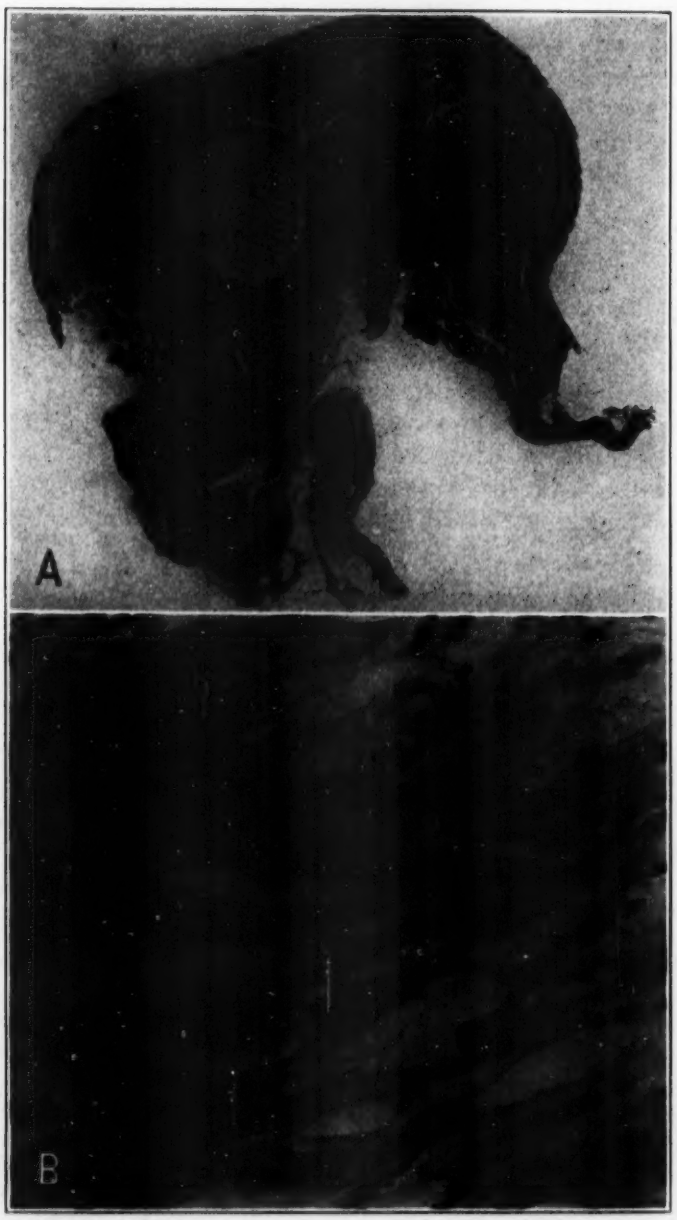

Fig. 1 (case 1).—*A*, low power photomicrograph showing the orbital portion of the optic nerve surrounded by tumor which fills the intervaginal space. The torn dura is an artefact. *B*, high power photomicrograph of the same section.

and stained with hematoxylin and eosin and with Mallory's connective tissue stain. A preliminary diagnosis of "benign fibrous tissue tumor" was made in each instance.

Autopsy observations of significance were limited to the cranial cavity. There were extensive hemorrhage and maceration of the base of the brain anteriorly. A small fragment of a yellowish gray tumor tissue was found in the region of the optic foramen. This was fixed in Helly's fluid and in a solution of formaldehyde and ammonium bromide.

Microscopically, all the specimens presented much the same picture (fig. 1 *B*). Save at the periphery, where a few degenerated nerve fibers were seen, the optic nerve was not remarkable. Surrounding this was a collar of dense tissue, which at first suggested a fibrous tissue tumor. Dense irregular bands of connective tissue divided the tumor into irregular areas, the picture resembling the coarsely reticulated type of Verhoeff.[11] The tumor cells were elongated, presenting small oval nuclei. Fine and coarse glia fibrils coursed irregularly through the mass. Many psammoma bodies were present. Sections impregnated with silver and gold stains were not entirely satisfactory but suggested a spongioblastoma polare.

The point of origin of the tumor in the nerve was not demonstrated, nor were the relations of the nerve to the intracranial portion of the tumor made entirely clear.

The case is registered at the Army Medical Museum (accession no. 50 514).

CASE 2.—*History.*—M. C. (fig. 2 *A*), a 3 year old Negro girl, was seen in the pediatric clinic on Jan. 11, 1939, because of unilateral exophthalmos of the right eye of eight months' duration. The family and past histories were entirely noncontributory. Two years before the child's entry the parents had noted an external strabismus of the right eye and sixteen months later a forward and downward displacement. The child had repeatedly complained of pain in the right eye for eight months, and vision seemed to be diminished. The day before the child's entry to the hospital bilateral swelling of the parotid glands developed, which prohibited admission to the hospital until February 2.

Examination.—Complete physical and neurologic examinations of the patient gave negative results except for the findings in the right eye. The left eye was normal. Visual acuity could not be tested. The right eye was displaced downward and outward; motility was greatly limited. The conjunctivas appeared normal. In the superior nasal quadrant there was a faint bulge in the sclera. Palpation revealed no mass or erosion of the orbital rim. The pupil reacted to light. The intraocular tension was normal.

On ophthalmoscopic examination the media were found to be clear. The disk could not be seen because of a large, grayish, elevated oval mass (fig. 2 *B*) which extended temporally to the macular region and about the same distance in other meridians. The mass appeared to be cystic and undulatory. There were no retinal vessels overlying it. These appeared to dip beneath the margin of the mass as they approached the disk. The arteries appeared larger than usual, the inferior nasal artery in particular being about twice the normal diameter. This artery was not accompanied by a vein, and about 4 disk diameters from the disk it suddenly dipped into the sclera. A short distance peripherally there was a small

11. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of Optic Nerve: A Histologic Study of Eleven Cases, Including Case Showing Cystic Involvement of Optic Disc, with Demonstration of Origin of Cytoid Bodies of Retina and Cavernous Atrophy of Optic Nerve, Tr. Sect. Ophth., A. M. A., 1921, p. 146.

area of old chorioretinitis. The remainder of the retina was normal except for the richness of the arteriolar coloring and the increased caliber of the walls of the vessels.

Roentgenograms of the optic foramens showed the right to be 9 mm. in diameter and the left 5 mm. Films of the skull and sinuses were normal.

Operation.—The day after the child's admission the globe and a large chocolate-colored, spindle-shaped tumor were removed, ether anesthesia being used. The

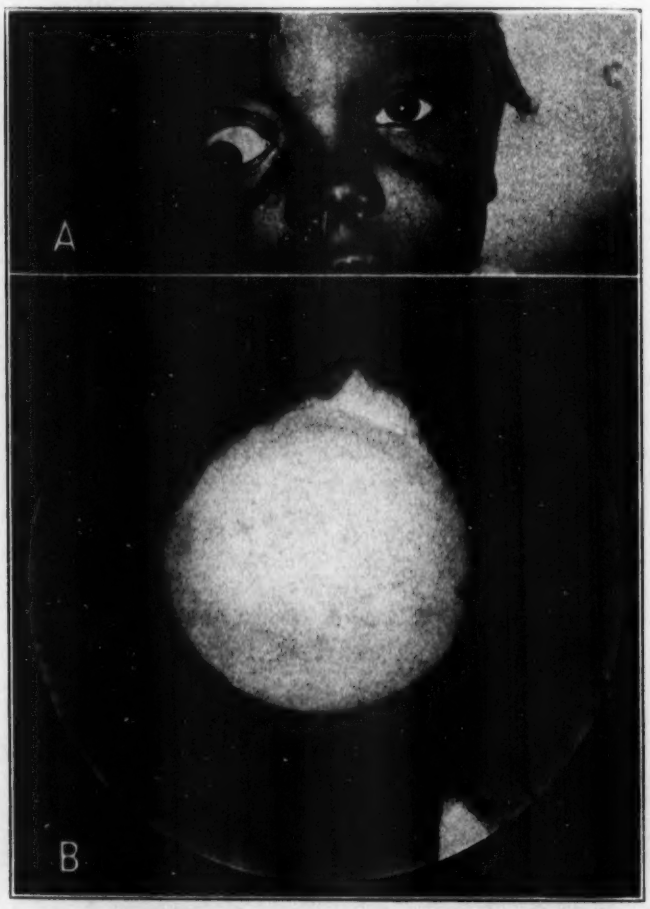

Fig. 2 (case 2).—*A*, photograph of the patient, showing the exophthalmos downward and laterally. *B*, photograph of a drawing of the tumor of the disk. Compare this with figure 3 *B*.

orbital portion of the tumor could not be felt until the globe was removed. This portion of the tumor measured 4 cm. in length and 1 cm. in breadth. The smaller end of the tumor extended into the apex of the orbit. No unusual bleeding was encountered.

The postoperative course was entirely uneventful, and the patient was discharged from the hospital on the twelfth day.

The patient returned for a check-up two months later. Roentgenograms of the skull were normal, and ophthalmoscopic examination gave negative results.

Pathologic Study.—The eyeball measured 2.5 cm. in the transverse diameter and grossly presented nothing remarkable. The optic nerve was cut level with the sclera and was not enlarged at this point. The eye was fixed in Bouin's fluid and sectioned after hardening.

The tumor was a sausage-shaped mass (fig. 3 *A*), measuring 3.6 cm. in length and 1.4 cm. in greatest diameter. At the larger end was a circular break of 1.5 mm. in the capsule, where it had been cut from its attachment to the eyeball.

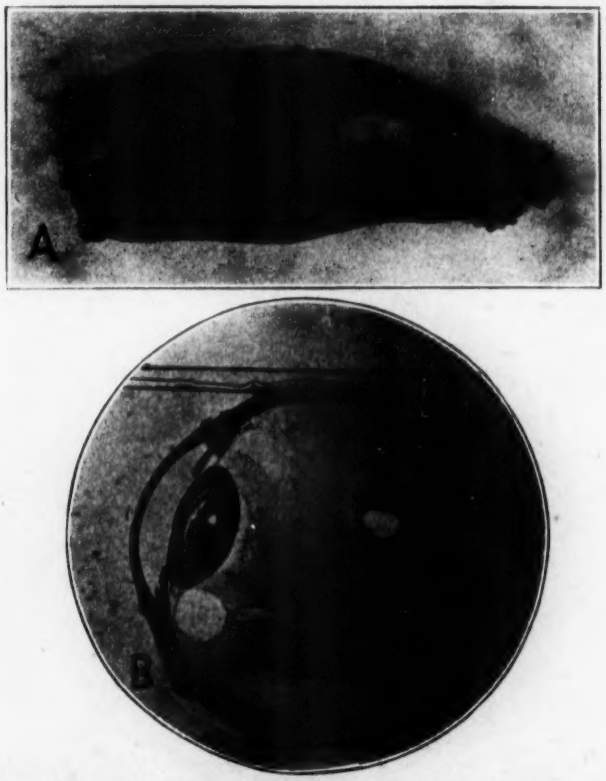

Fig. 3 (case 2).—*A*, gross appearance of the intraorbital tumor. The small teatlike process at the left is the point of attachment to the bulb. *B*, section through the globe; × 2. The photograph was overexposed to bring out the areas of liquefaction in the vitreous and to demonstrate the fibrinous exudate on the surface of the tumor. Compare this picture with figure 2 *B*.

At the opposite end the thin capsule had been cut through, and the myxomatous tumor tended to extrude. Grossly, it appeared to have been incompletely removed. The cut surface presented a soft, translucent, whitish appearance in which a few longitudinal striations were noted. The tissue was fixed in solution of formaldehyde, Zenker's fluid and in a solution of formaldehyde and ammonium bromide. Sections were stained with hematoxylin and eosin, Mallory's connective tissue and phosphotungstic acid stains and Bielschowsky's silver stain and were impregnated with silver carbonate and gold chloride sublimate.

The mass of the tumor was composed of a loose reticulum of glial tissue, separated into irregular bundles by thick bands of tissue, in part fibrous and in part compressed glial material (fig. 4 *B*). The loose portion consisted largely of glia fibers, but many cells were noted, the oval nuclei of which contained a faint

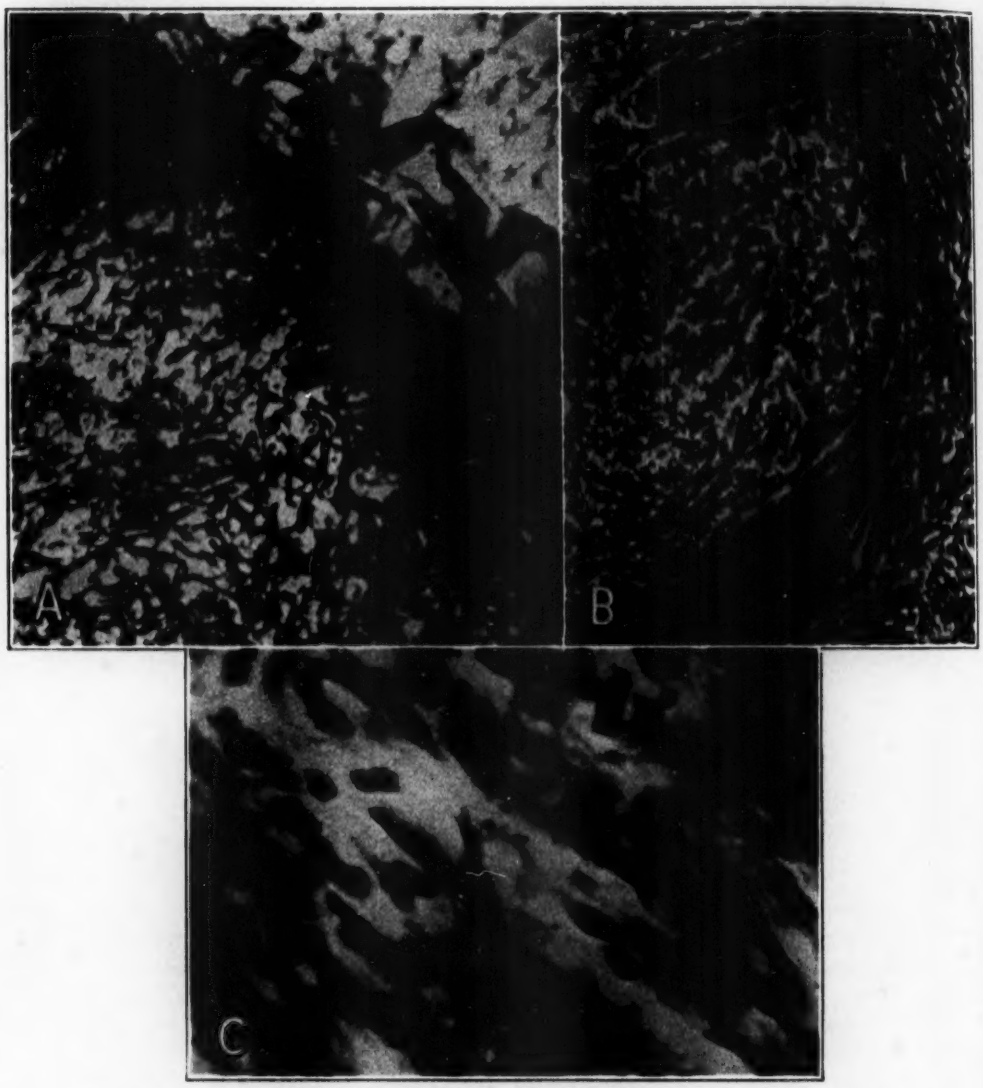

Fig. 4 (case 2).—*A*, section through the edge of the intraocular portion of the tumor. Mallory's connective tissue stain, × 140. Note the looseness of the tumor, the astrocytes and spongioblast and the fibrinous exudate at the periphery of the tumor. *B*, intraorbital tumor. Hematoxylin and eosin stain, × 140. Note the fibrous septum dividing the looser tumor into irregular spaces filled with glial cells and fibrils. *C*, intraorbital tumor. Fixation with solution of formaldehyde and ammonium bromide and silver carbonate impregnation, × 650. Note the elongated spongioblasts.

chromatin network and several (frequently two) sharply defined chromatin dots. The cells possessed little cytoplasm, which occasionally could be seen prolonged into the glial processes. Unfortunately, no myelin stains were made, but Bielschowsky's silver stain did not reveal any axis-cylinders, nor did the hematoxylin and eosin stain suggest the presence of any nerve fibers. The gold and silver impregnations (fig. 4 *C*) showed the majority of the cells to be polar spongioblasts, though a few plump astrocytes were present.

Microscopic study of the eye revealed little of importance save the tumor. The vitreous (fig. 3 *B*) presented several cystlike areas of liquefaction. Rising from the surface of the tumor mass, numerous fibrinous strands extended anteriorly, but no actual invasion of the vitreous by tumor was noted. The hyaloid membrane was intact save over the summit of the tumor, where its outline was lost in the fibrinous material.

The disk was swollen and elevated, being 1.5 mm. in thickness. It was surmounted by a flattened mushroom-like mass of the same thickness, lying so close that at first glance it seemed to be part of the disk. However, microscopically there was seen a dividing line, formed by a reflection of the internal limiting membrane.

When the tumor was approached from the normal retina at the periphery, the rod and cone layer and the external nuclear layer were first involved, being thinned and compressed by the tumor growth. The outer reticular layer was fairly well preserved. The inner nuclear layer gradually became disorganized, showing microscopic cystlike spaces. The inner reticular and ganglion cell layers were completely disorganized. The internal limiting membrane was thrown into a series of rounded serrations, which flattened out as the slope of the tumor was ascended. In the deeper portion of the tumor, the disk, there was a large amount of fibrous tissue intermingled with the glia fibers. The upper mushroom-like portion consisted almost entirely of glia, most of the cells appearing to be bipolar spongioblasts (fig. 4 *A*). Many astrocytes were likewise noted. No cysts were present as in the other cases reported.

COMMENT

Since the optic nerve is not a nerve in the true sense of the word but rather an extension of the brain substance, even to its fibrous sheath, and since the studies of Marchesani [12] have shown that the same neuroglial elements are present as are found in the brain, it is only logical to suppose that the glial and fibrous tissue tumors of the nerve should be comparable to those of the brain. There are now available the histologic technics of Cajal and del Rio-Hortega, which were not available when Verhoeff [11] offered his microanatomic classification in 1921 (finely reticulated, coarsely reticulated and coarsely fibrillated), and the trend toward a specific qualitative analysis of these tumors is evident in the recent literature. Mehney [13] reported 3 cases of spongioblastoma

12. Marchesani, O.: Die Morphologie der Glia im Nervus opticus und in der Retina, dargestellt nach den neuesten Untersuchungsmethoden und Untersuchungsergebnissen, Arch. f. Ophth. **117**:575, 1926.

13. Mehney, G. H.: Primary Tumor of Optic Nerve: Report of Case, Arch. Ophth. **16**:95 (July) 1936.

polare; Kiehle,[14] Stern [15] and Rand, Irvine and Reeves [16] each reported 1 case. DeLong [17] implied that the growth in his case was of this type. Goldstein and Wexler [18] reported a case of spongioneuroblastoma and Santori [19] a case of astrocytoma. Eickhoff,[20] Foerster and Gagel [21] and Seefelder [8] reported cases of spongioblastoma multiforme. Lundberg [7] reported 9 cases of oligodendrocytoma, and Schreck [22] and Cárdenas [23] each reported a case.

Since the term glioma no longer suffices, the differentiation into specific types of glioma must be attempted. It is felt that while many of the tumors can be accurately diagnosed by the usual glial stains, the best, and in many cases the only accurate, technics are the impregnation methods of Cajal and del Rio-Hortega. These require special fixation in a solution of formaldehyde and ammonium bromide and experienced technical preparation, their final interpretation being based on the predominating cell type. Also, it may be noted that in our experience Mallory's phosphotungstic acid–hematoxylin stain for glia is reliable only after fixation in Zenker's fluid. Thus, of the 23 cases cited, only in that of Cárdenas [23] are the impregnation methods known to have been used, while in several others only tissue that had been fixed in solution of formaldehyde was available. We emphasize, therefore, that while there is need for a specific differentiation of these tumors so that a rational prognosis can be made (since the various types present

14. Kiehle, F. A.: Tumor of Optic Nerve: Report of Case, Arch. Ophth. **15**:686 (April) 1936.

15. Stern, R. O.: Tumor of Optic Nerve, Chiasma and Thalamus, Proc. Roy. Soc. Med. **30**:1096, 1937.

16. Rand, C. W.; Irvine, R., and Reeves, D. L.: Primary Glioma of the Optic Nerve: Report of a Case, Arch. Ophth. **21**:799 (May) 1939.

17. DeLong, P.: Primary Tumors of the Optic Nerve: Report of Case, Am. J. Ophth. **17**:797, 1934.

18. Goldstein, I., and Wexler, D.: Spongioneuroblastoma of Optic Nerve in Neurofibromatosis (Recklinghausen), Arch. Ophth. **7**:259 (Feb.) 1932.

19. Santori, G.: Glioma primitivo astrocitario diffuso del nervo ottico, Boll. d'ocul. **9**:464, 1930.

20. Eickhoff, W.: Intraneurales Wachtsum eines Glioms (Nerv. opticus), Virchows Arch. f. path. Anat. **302**:222, 1938.

21. Foerster, O., and Gagel, O.: Ein Fall von sog. Gliom des Nervus opticus—Spongioblastoma multiforme ganglioides, Ztschr. f. d. ges. Neurol. u. Psychiat. **136**:335, 1931.

22. Schreck, E.: Ein Oligodendrogliom als Beitrag zu den primären intraduralen Optikustumoren, Klin. Monatsbl. f. Augenh. **100**:560, 1938.

23. Cárdenas Pupo, M. D.: Contribución al estudio del oligodendrocitoma del nervio visual (Río-Hortega), Bol. Liga contra el cáncer **9**:196, 1934.

various degrees of malignancy), the methods by which this differentiation is reached must be carefully evaluated.

Invasion of the disk by a tumor growth of the optic nerve is rare, only 15 cases being reported so far in the literature. And although the statement has been made that meningiomas more frequently involve the disk than gliomas, only 6 cases of the former are found but, with the 1 case here reported, 10 cases of the latter are recorded.

Invasion by meningioma has been reported by Jacobson,[24] Goldzieher,[25] Baeumler,[26] Neame,[27] Schwarz [28] and Coston.[29] Hirschberg's case,[30] which is occasionally cited, was probably one of sarcoma of the choroid, although von Recklinghausen thought that the growth arose from the disk.

Of the gliomas, von Graefe's case [31] in 1864 is probably the first recorded. Barraquer's case [10] is especially interesting in that the patient was followed for nine years, and clinically the growth showed intracranial extension. The case of Sulzer and Rochon-Duvigneaud [32] was probably one of glioma, while that of Pereyra [33] in 1914 undoubtedly was. Verhoeff,[11] Martin and Cushing,[34] Sattler [35] and Weigelin [36] reported cases in 1921, 1923, 1926 and 1931, respectively, the growth in Sattler's case being extraordinarily like that described here (case 2). Finally, using more specific terminology, Foerster and Gagel [21] in 1931

24. Jacobson, P. J.: Klinische Mittheilungen, Arch. f. Ophth. (pt. 2) **10**:55, 1864.

25. Goldzieher, W.: Die Geschwülste des Sehnerven, Arch. f. Ophth. (pt. 3) **19**:139, 1873.

26. Baeumler, E.: Ein Fall von Orbital- und Uvealsarkom, Klin. Monatsbl. f. Augenh. **24**:5, 1886.

27. Neame, H.: Tumour of Optic Nerve, Brit. J. Ophth. **7**:209, 1923.

28. Schwarz, K.: Zur Klinik und Anatomie der Geschwülste des Sehnerven, Arch. f. Ophth. **135**:247, 1936.

29. Coston, T. O.: Primary Tumor of Optic Nerve, with Report of Case, Arch. Ophth. **15**:896 (April) 1936.

30. Hirschberg, J.: Casuistische Mittheilungen über Geschwülste der Orbita und des Bulbus, Klin. Monatsbl. f. Augenh. **6**:153, 1868.

31. von Graefe, A.: Geschwülste des Sehnerven, Arch. f. Ophth. (pt. 1) **10**:193, 1864.

32. Sulzer and Rochon-Duvigneaud: Néoplasme du nerf optique et de la papille, Ann. d'ocul. **149**:161, 1913.

33. Pereyra, G.: Mixoma del nervo ottico, Ann. di ottal. **43**:456, 1914.

34. Martin, P., and Cushing, H.: Primary Gliomas of Chiasma and Optic Nerves in Their Intracranial Portion, Arch. Ophth. **52**:209 (May) 1923.

35. Sattler, H.: Die bösartigen Geschwülste des Auges, Leipzig, S. Hirzel, 1926, p. 238.

36. Weigelin: Ueber Gliomatose des Sehnerven, Klin. Monatsbl. f. Augenh. **87**:527, 1931.

reported a case of spongioblastoma multiforme of the intracranial portion of the nerve which grew peripherally and was seen at the disk with the ophthalmoscope.

It is noted that no distinction is made as to the exact point of origin of these tumors in the nerve; we doubt that any of the gliomas arose primarily in the disk within the eye; but this, of course, is a matter of speculation. Should they arise there, however, one would first expect visual symptoms and a tumor visible at the disk long before the development of exophthalmos. In Stallard's case [37] (a primary neurofibroma of the disk) the first symptom was failing vision, the tumor being confined entirely to the disk within the eye.

The most important question, however, is that of treatment. With reference to the meningiomas, the answer is not difficult. These tumors tend to recur; they tend to extend centrally; and no case of distant metastasis is recorded. It would seem, therefore, that the procedure of choice in such cases is craniotomy, followed, if necessary, by exenteration of the orbit or some less radical procedure.

In the second group, that of the glial tumors, the problem is not so simple, and a disagreement exists between us as to the procedure of choice. This type of tumor does not tend to metastasize even locally but rather extends centrally by way of the nerve, contained entirely within the sheath. In at least 1 case [10] long delayed intracranial involvement is recorded, although in 4 others incomplete removal was not followed by recurrence in periods ranging from seven to seventeen years.

In our second case craniotomy was not done. In Penfield's excellent book,[38] Verhoeff made the following statements concerning the gliomas:

> Within the nerve stem the growth does not advance by invading the original structure, but by causing the pre-existing neuroglia in the vicinity of the tumor to proliferate and take on the character of the tumor tissue. This would seem to indicate that some substance is produced by the tumor which stimulates the contiguous neuroglia.

And again:

> The possible practical importance of this observation lies in its indication that removal of the largest part of the tumor may do away with the assumed stimulating substance and thus prevent further extension of the growth.

Certainly cases such as those previously cited seem to bear out at least the principle of this concept, viz., that simple removal by the anterior approach through the orbit may result in cure. On the other hand, these are isolated instances, and a study of such a large group

37. Stallard, H. B.: Case of Intra-Ocular Neuroma (von Recklinghausen's Disease) of Left Optic Nerve Head, Brit. J. Ophth. **22**:11, 1938.

38. Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1035.

of cases as Hudson [9] collected shows how inadequately many were studied. On the basis of his 118 collected cases of primary glioma of the optic nerve, the following analysis is made:

Death from meningitis	10 cases
Death from recurrence	6 cases
Probable recurrence	5 cases
Six year cure	6 cases
One year cure	34 cases
Patients followed less than one year	57

Of the cases in which the patients were followed less than one year, constituting 48 per cent of the cases, we know nothing. And in only 5 per cent of the cases was a six year cure recorded. In the face of these findings, it is impossible for the author on the pathologic side (J. M. W.) to fit into his general concept of tumor growth such a view as is held by Verhoeff,[11] viz., that partial removal may be followed by a cure. He believes with Gluck [39] that it is a preponderance of the slower growing types that accounts for the relatively long survival after incomplete removal. He therefore advocates primary craniotomy (which he believes is mandatory in those cases in which enlargement of the optic foramen is seen roentgenographically), followed by a second operation from the front for removal of the orbital portion of the tumor which remains. He believes that the problem is one for the neurosurgeon rather than for the ophthalmologist. That there has been a general tendency in this direction since Dandy's paper [2] in 1922 is noted by the case reports of Mehney [13] and of Rand and his co-workers.[16] In the latter case the tumor growth extended through the foramen (enlarged by roentgen examination) to a point 1.5 cm. from the chiasma.

That this procedure is too radical may be a just criticism, in view of the long survival in some of the cases cited. The risk is admittedly great. In the 5 cases recorded, including our own case 1, 2 of the patients died of the operation, the growth in 1 case being a primary intracranial tumor (Mehney's case 3). Yet in view of what may be accomplished in some cases, it is though that the chance of operative fatality is outweighed by the chance of a more likely permanent cure than is offered by the simple removal within the orbit.

SUMMARY

1. Two cases of primary spongioblastoma of the optic nerve are reported, one showing the unusual picture of extension into the optic disk.

39. Gluck, B.: Glioma of the Optic Nerve, Brit. J. Ophth. **16**:406, 1932.

2. The need for complete pathologic study of these tumors is stressed, so that by accurate differentiation a more rational prognosis can be made. It is felt that the impregnation methods of Cajal and del Rio-Hortega offer the best means toward this end.

3. Although the operative mortality after craniotomy in the 5 recorded cases is 40 per cent, analysis of the cases reveals a much brighter prognosis, and the neurosurgical approach is advocated by one of us (J. M. W.).

ORTHOPTICS AT THE CROSSROADS

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BROOKLYN

It is only within recent years that the scientific basis for binocular training was advanced and applied by a small group of serious workers, even though some form of exercise for the eyes has been prescribed empirically for many years. Unfortunately, the principles and methods that they fostered won the sympathy of but few ophthalmologists. During the last decade, with its development of instruments for ocular training, there has been on the one hand an unfortunate "groping in the dark" regarding orthoptics and on the other hand an unfriendly skepticism by those too accustomed to established clinical methods, with the result that the favorable aspects of scientifically controlled orthoptic training have been largely obscured. The interest in orthoptic training has not progressed substantially in recent years, and one must reflect as to whether this trial period has been sufficient to evaluate its merits as a therapeutic aid and whether it should be left to proceed on the feeble momentum generated by the sporadic efforts of a few ophthalmologists or be worthy of a more general interest.

A number of British investigators as well as a few in this country have over an extended period carried out studies on various types of binocular imbalance and have discovered distinct benefits in properly selected cases from binocular training carried out in a systematically controlled manner. And yet these reports have not generated interest in controlled orthoptics. I believe that the successful practice of orthoptics presents difficulties that are overwhelming to the average ophthalmologist, and a plan is proposed to ease the responsibilities of the practitioner who would still prefer to supervise personally the management of his cases of binocular imbalance.

The difficulties in controlled orthoptic training are: (1) the matter of expense and (2) the management of the child.

The cost of training based on numerous sittings usually discourages most patients, especially when the length of such treatment cannot be foretold. In my practice I have had the patient purchase or rent a suitable binocular instrument for home use, after a few satisfactory office sittings, in order to save the expense of further frequent visits. Unfortunately, even such an instrument is too expensive for the average patient.

Moreover, the patient has to return every week or two for periodic examinations to determine progress made with binocular or orthoptic

training and for further recommendations as to exercises. This item of expense is not considerable.

Home training requires an intelligent and cooperative parent or guardian who has to understand the purpose of these exercises, the method of exercising on a binocular instrument, how to hold the child's interest, what pictures to use, how long and how often to carry out such training and the necessity of having the child, whenever possible, keep systematic records of his accomplishments; these and many other details demand the cultivation of a specialized interest and training on the part of the ophthalmologist.

The problem of caring for cross eyes and binocular imbalances in preschool children as well as in children in the junior grades cannot be tackled by the ophthalmologist alone. During this formative age period, in which operations are generally avoided, conservative measures, including controlled binocular training, are often advisable in order that possible good may be done to the child. And even when operations are found necessary, these same conservative measures may have to be supplemented in order to maintain the benefits for which the operations were intended. In my opinion, it is only by concerted efforts of both parents and school authorities that such cooperation may be effected. This can be accomplished in two ways: by publicizing the need for early and careful attention to cross eyes and eyestrains and by a simple, practical comprehensive plan for teamwork between the physician and the teacher or the psychologist.

The health teacher or health education department of the school can further the solution of the problem of cross eyes by including among its health gospels such simple teachings as:

1. Early treatment of cross eyes by glasses or by exercises can often correct such a condition. Operation is necessary only when other measures have failed; moreover, it is devoid of danger.

2. Children do not usually outgrow cross eyes.

3. Cross eyes is not a condition to be laughed at but one requiring careful attention to the eyes. (Teachers can do much to counteract the jibes of classmates by keeping photographic posters showing cross eyes before and after treatment.)

4. An eye which has become crossed and is left alone is often likely to turn into a blind eye.

The management of squint is more than a medical or a surgical problem. It is also a psychologic problem,[1] both in regard to the need for a sympathetic understanding of the inferiority complex of the afflicted

1. Krimsky, E.: Psychologic Considerations in the Study of Binocular Function, Arch. Ophth. **21**:662-670 (April) 1939.

child and in regard to its correction through nonoperative means, for instance by orthoptic training. However expert the ophthalmologist may be in the surgical or cosmetic correction of squint, comparatively few are at present sufficiently trained or patient enough to administer ocular exercises to the cross-eyed child without assistance. And the assignment of such a child to an orthoptist for supervision and control creates a barrier which neither the ophthalmologist nor the technician desires.

In my opinion, the ophthalmologist who recommends binocular training should either assume personal management of his patient or assign him to another colleague trained in such procedure rather than to a technician. The trained ophthalmologist can evaluate instrumental with clinical findings, whereas the orthoptist thinks primarily of instrumental responses and is likely to attach too much academic importance to detailed readings. In order that orthoptics can be practiced successfully, the ophthalmologist must become as familiar with binocular study as with refraction. He alone should determine whether his patient has an abnormal or a normal retinal correspondence and should be able to ascertain the binocular status accurately. With such a clinical and instrumental record carried out periodically, if necessary, he can better judge the true significance of orthoptic training without actually becoming involved in its detailed application.

With the present economic setup, few children can avail themselves of orthoptic training in a satisfactory manner. In a large city like New York there are only three orthoptic clinics, and these are either unavailable or inaccessible to the large majority of needy children. With such insurmountable difficulties, the cross-eyed child must turn to the school as his haven for binocular training. In recent years the management of other defects of children has been shared by the school system. Among these one may mention: speech difficulties (a psychologic problem), deafness (detected by audiometric tests), cardiac conditions and behavior problems. The training of a child with binocular imbalance includes more than the mere purchase or rental of an instrument; it is a psychologic problem to be studied by one familiar with the peculiar mental makeup of the individual child, and for selected cases of binocular imbalance the school teacher can best attend to such a function without interfering to any appreciable extent with her duties as teacher or with the child's program of work and play. Nor need the teacher be specially trained for such a project, except to have the child report to the ophthalmologist for periodic checkups and for further instructions, which can be written in concise, simple language. The teacher as psychologist may serve as the natural complement to the ophthalmologist as clinician in the correction of squint or binocular imbalance with normal retinal correspondence in selected cases.

CONCLUSIONS

1. The problem of caring for the cross-eyed child from the conservative standpoint is a vast one that cannot be solved by the ophthalmologist alone. It is one that requires an intimate cooperation with the school system. The nursery school or kindergarten provides a sympathetic environment for the young child.

2. Any makeshift or haphazard approach to orthoptic training will justify the criticism of ophthalmologists regarding a method which has till now been looked on with some indifference.

3. It is unfortunate that at the present time but a small number of ophthalmologists are qualified to treat the subject in a critical, analytic manner. It is on the teachings of these men that younger, serious workers will have to depend to spread an approved scientific management of binocular imbalances.

4. Not all ophthalmologists will agree to such a program. As in other fields of human endeavor, one must expect different schools of thought and practice, each with its particular merits; but there must be sufficient representation and serious interest among these competing schools to reduce the incidence of squint and imbalances.

Clinical Notes

ANGIOMATOSIS RETINAE

EDWARD P. RYAN, M.D., NEW YORK

B. T., a 13 year old Jewish boy, was first seen in Dr. Fletcher's clinic at the Manhattan Eye and Ear Hospital on March 4, 1939, with the complaint of failing vision in the left eye for about one year and a half. This impairment of vision was discovered in a routine examination of the eyes at school, but no notice was taken of it by the patient or his family. In my routine examination of the fundi

Drawing of the left fundus of the patient.

an abnormality of the blood vessels was discovered only in the left eye. The vision was 20/20 in the normal right eye. Vision in the left eye was limited to the counting of fingers at 1 foot (33 cm.). Externally every structure was normal in appearance. The pupil reacted to light and in accommodation. The media were clear throughout. The fundus presented an unusual picture of immense, dilated, tortuous and distorted "intestine-like" vessels covering the papilla and the immediate surroundings, particularly the temporal side, as shown in the accompanying drawing. No cystlike formation or structure was found at the periphery of the fundus. There were no exudates, hemorrhages or retinal detachment. Physical examinations, including neurologic, roentgenologic, and otolaryngologic consultations, gave essentially negative results, except for diseased tonsils.

Read before the Section of Ophthalmology of the New York Academy of Medicine, May 15, 1939.

The boy was apparently in good health and still is. His past history and family history were noncontributory. There had been no trauma at any time.

In the case just described the interest is in the tremendous size and the marked tortuosity of the retinal vessels. In view of the rarity of this condition and a diversity of diagnoses, ranging from angiomatosis retinae to aneurysma racemosum arteriovenosum, as expressed by several leading ophthalmologists, this case, though titled "angiomatosis retinae," might be better classified as a case of abnormality of the retinal vessels, probably of a congenital nature.

KELOID OF THE CONJUNCTIVA

Report of a Case

NORMAN P. SCALA, M.D., WASHINGTON, D. C.

M. A. C., a white woman about 37 years of age, was referred to me by Dr. George W. Creswell on July 26, 1938. She stated that in March 1920, while she was working for the government in Boston, a hemorrhage occurred in her right eye. She was referred by the government authorities to Dr. H. B. C. Riemer, an oculist of Boston, who made a diagnosis of ecchymosis of the bulbar conjunctiva on the temporal side of the right eye.

From that time on, the patient stated, her right eye gave her trouble. She complained of photophobia, severe headaches, the sensation of having a foreign body in her eye and a feeling that her eye was crossed. The left eye was entirely normal. The right eye was normal except for what looked to be a cystoid degeneration of the conjunctiva on the temporal side just posterior to the insertion of the external rectus muscle.

When the patient was sent to me, it was thought that she had a foreign body in her eye. However, a thorough examination did not reveal the presence of one.

Several attempts were made to remedy the condition by superficial removal of the tissue, but after each attempt it only became more aggravated, and the mass increased in size after each operation.

It therefore became necessary to hospitalize the patient and remove the affected tissue down to the sclera, including some of the muscle fibers. The tissue was removed well outside the affected area; the denuded surface was covered with healthy conjunctiva from above and below, and four interrupted sutures were inserted. The patient was hospitalized from September 12 to 26 and was unable to return to work until October 17.

The tissue was sent for examination to Col. J. E. Ash,[1] of the Army Medical Museum, who made the following report:

"The specimen from your patient, M. A. C., our accession no. 63668, is a minute piece of firm fibrous tissue showing nothing significant grossly.

"Microscopically the specimen is partially covered by conjunctival epithelium, just beneath which there are foci of edema and congestion but very little exudate. The bulk of the mass consists of dense collagenous bundles containing few elongated and narrow nuclei, groups of blood vessels which have the appearance of being

1. Curator, Army Medical Museum, Washington, D. C.

the remnants of granulation tissue, several thick-walled and older vessels and some small mononuclear exudate scattered about the younger blood vessels. I can see no neoplasm and would interpret the picture as quite hyperplastic scar tissue showing a keloid tendency."

COMMENT

Since the removal of the tumor formation and the healing of the wound, the patient stated that all of her symptoms have abated and that she is able to continue her work with comfort and without glasses. It is probable that this condition started in 1920 when she had the hemorrhage, which was probably due to the strain the eye was under from the great amount of work she was compelled to do and the long hours required to do it. The symptoms complained of afterward were, no doubt, the result of scar tissue which formed in the region of the external rectus muscle and involved some of its fibers, thereby causing a limitation of the movement of the muscle. The removal of this mass together with the muscle fibers gave the eye more play and resulted in an abatement of the symptoms, for which the patient was grateful.

SUMMARY

A case of keloid of the conjunctiva with marked subjective symptoms is reported. Clinically the picture was that of cystic degeneration of the conjunctiva and was indistinguishable from that of the more common type of such degeneration.

From a review of the literature, it is evident that a keloid of the conjunctiva is a rare condition, as this is the first case that could be found that has been reported. There has been reported, however, to the Eye Registry, Army Medical Museum (accession no. 38976) an instance of keloid of cornea [2] by Dr. Henry C. Smith, of Nashville, Tenn., and I understand that he is preparing a report of this case for publication.

2. Smith, H. C.: Keloid of Cornea, to be published.

SPECTACLE LENSES FOR USE DURING OPERATIONS

GEORGE M. RICHARDS, M.D., STONY POINT, N. Y.

To overcome some of the disadvantages presented by the use of the Beebe loupe and its modification during an operation, especially when the operator is presbyopic or desires a comfortable working focus of 15 to 20 cm., the following bifocal lens was designed.

The essential advantage of this lens is that a working area is afforded in the upper field as well as in the lower, and at the same time free lateral vision is permitted. To accomplish this a segment 10 by 24 mm. is placed almost vertically near the middle of an ordinary spectacle lens and equidistant from the top and the bottom of the lens. The upper pole of each segment is tilted templeward 1 mm. to allow for separation of the visual axes in the upper fields. The segments are

centered for the operator's pupillary distance when his eyes are focusing at 15 to 20 cm. A 1 degree prism, base in, is ground in each segment to relieve some of the excessive convergence at this short focus. The lens on which the bifocal segment is placed can have the operator's distance correction or an intermediate correction to focus at about 60 cm., which is convenient for reaching and selecting instruments. The strength

Spectacle lenses for use during an operation.

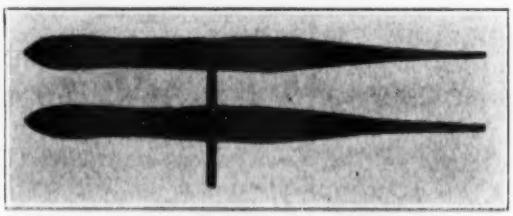
of the bifocal lens will average near a + 3.00 addition to the intermediate correction or a + 4.00 addition to the distance correction. The segments are cemented in place. If a pad-bridge frame is used, large (jumbo) pads are recommended to distribute better the extra weight of the frame and lenses.

This design, shown in the accompanying sketch, is submitted in the hope that it will help others who have had similar difficulties.

BIPOLAR FIXATION

ROBERT R. BLONDIS, M.D., CLEVELAND

The desirability of bipolar fixation of the globe during surgical procedures has frequently been mentioned. This is usually obtained with the aid of an assistant. The forceps pictured here obviate the necessity for an extra hand in the operative field, since pressure on



Bipolar fixation forceps.

one forceps operates also the other and gives satisfactory bipolar fixation. The instrument consists of two ordinary fixation forceps, which can be locking or nonlocking in type, on a sliding bar. The distance between them can be varied and exactly maintained. A measured scale on the bars can be used for the exact resection of muscle or other tissue. More individual forceps can be placed on the double bars and used to approximate incisions in the skin and other structures evenly.

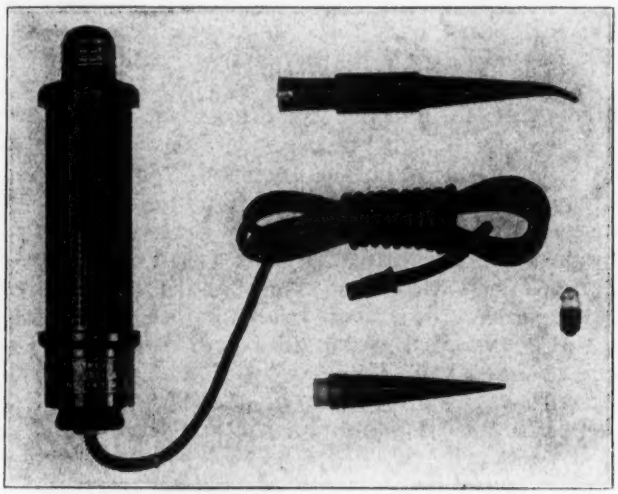
OCULAR TRANSILLUMINATORS MADE OF LUCITE

HAROLD M. BLOCK, M.D., DALLAS, TEXAS

The transilluminators described here are not new in the field of ophthalmic instruments. They are a modification of those made by a European manufacturer of instruments.

Since lucite [1] has optical properties similar to those of quartz, it lends itself well to the transmission of light. It has the disadvantage, however, of being destroyed or injured by high temperatures and various chemicals, but it is easily molded and worked with.

The transilluminators described here are each approximately 3 inches (7.6 cm.) long and taper from 13 mm. at their base, where light is admitted, to 1.5 mm. at its point of exit. One of the transilluminators is



Ocular transilluminators made of lucite.

straight; the other has a slight curve, the light emerging at an angle of approximately 45 degrees. The straight transilluminator is adapted for examination of the more anterior parts of the globe, and the curved one is adapted for inspection of the deeper recesses in the cul-de-sacs. With the curved type, light is thrown toward the interior of the globe through the sclera, which of course is impossible with a straight transilluminator. The curved transilluminator is silvered on its outer surface and then opaqued, but the straight one is only opaqued.

The light source is a magnifying globe of the flashlight type, with a double filament. It operates on a variable current of from 6 to 7 volts.

From the Department of Ophthalmology, Baylor University College of Medicine and Baylor University Hospital.

Mr. Don Cameron, of the Cameron Surgical Specialty Company, lent technical aid.

1. Lucite is a du Pont product (methyl metharcylate).

This globe gives out an intense beam of light, which is picked up and concentrated through the small opening at the exit of the transilluminator and measures 2½ foot candles on a current of 7 volts. A rheostat is available so that the intensity of the illumination can be varied.

Owing to the physical properties of lucite and the opaquing process, the solutions for sterilization are limited. These four may be used: potassium mercuric iodide (Parke, Davis & Co., germicidal disks), 1 tablet in 4 ounces (120 cc.) of water, freshly prepared; mercury bichloride, 1:400 solution; hexylresorcinol, 1:1,000 solution, and a solution of merthiolate. I have used hexylresorcinol, as I do not believe it will damage the metal parts of the transilluminator as do the metallic salts or iodine. However, the instruments are rarely, if ever, used in actively infected eyes, and sterilization under these circumstances is not as difficult.

My own experience with these instruments has proved highly satisfactory, and I am well pleased. The only disadvantage is that the light in the handle gets warm after three to five minutes' usage, but this is not a serious drawback, since the tip of the transilluminator, that part in contact with the eyeball, remains cool. A local anesthetic of one of the cocaine substitutes is sufficient when these instruments are employed. Patients react well, as there is no pain, heat, glare or pressure.

There are already several ophthalmic transilluminators on the market, but in designing these I paid particular attention to the exit point of light. If a tumor is small, say 1 to 2 mm. in size, and the opening larger than the tumor itself, the transmission of light will not be inhibited. Since the point of exit of light in these transilluminators is about 1.5 mm., I feel that in event of a small tumor or foreign body the transmission of light will be inhibited, making the contrast more noticeable. With the tip of the opaqued transilluminator against the globe, there are no blinding or confusing rays of light in a completely dark room. The observer, then, can get the maximum contrast from all accessible areas of the sclera.

513-514 Medical Arts Building.

News and Notes

EDITED BY W. L. BENEDICT

UNIVERSITY NEWS

Postgraduate Course in Aviation Ophthalmology and Aviation Medicine.—A postgraduate course in aviation ophthalmology and aviation medicine for graduates in medicine will be given by the George Washington University School of Medicine, Washington, D. C., from April 1 to 6 inclusive. This course is part of the postgraduate course in ophthalmology given from March 19 to 30 inclusive.

Course I is a special practical course in ocular surgery, pathology and orthoptics, limited to 25 participants, March 19 to 23 inclusive; course II, an intensive postgraduate course in ophthalmology, March 25 to 30 inclusive, and course III, a postgraduate course in aviation ophthalmology and aviation medicine, April 1 to 6 inclusive.

Among the guest lecturers are 17 officers of the Medical Corps of the U. S. Army and the U. S. Navy and Civil Aeronautics Authority. Credit will be given by the War Department and the Navy Department to those officers in the Medical Reserve Corps of the U. S. Army and the U. S. Navy who take course III. The fee for this course will be $40. The course will be given under the direction of Dr. W. Thornwall Davis, professor of ophthalmology at George Washington Medical School, assisted by the members of the staff of the department of ophthalmology.

Course in Visual Optics and Physiology.—The Harvard Medical School will give a course in visual optics and physiology from July 1 to July 27, 1940. This course, conducted by Drs. Ludvigh, Cogan and Easton with the assistance of other members of the staff, will be an all day course given six days a week during the month of July. Lectures, laboratory work and discussion will occupy seven hours a day, and outside reading will be assigned covering articles on special subjects in the modern American and English literature. The first two weeks of the course will cover approximately the range of material given in Duke-Elder's "Textbook of Ophthalmology" (vol. 1, pp. 483 to 1086). Subsequently, a treatment of more advanced topics not thoroughly discussed in the average English or American textbook will be undertaken.

PERSONAL

Dr. R. Townley Paton has been appointed professor of ophthalmology and head of the department of ophthalmology at the New York Medical College. Dr. Edwin S. Munson, who preceded Dr. Paton, has been retired for three years. The old New York Ophthalmic Hospital has been recently merged with the college. Dr. Paton will retain his position as surgeon at the Manhattan Eye and Ear Infirmary but will devote most of his time to the reorganization of the department of ophthalmology at the New York Medical College.

Dr. Charles M. Swab has been appointed director of the department of ophthalmology at the Creighton University School of Medicine. Dr.

Swab graduated from Creighton University in 1916 and received an M. S. degree in medicine from the University of Pennsylvania in 1934. He has been associated with the department of ophthalmology in the School of Medicine and the Creighton Memorial St. Joseph's Hospital in the capacity of associate professor since 1932.

Dr. Louis B. Bushman, who has been director of the department since 1919, has been made professor emeritus.

SOCIETY NEWS

Oxford Ophthalmological Congress.—The Oxford Ophthalmological Congress will be held at Oxford, England, from July 4 to 6, 1940. Prof. W. E. Le Gros Clark will deliver the Doyne Memorial Lecture, and there will be a discussion on "Emergencies and Complications of the Operation for Cataract."

Ophthalmological Society of the United Kingdom.—The Annual Congress of the Ophthalmological Society of the United Kingdom will be held in London, England, at the Royal Society of Medicine on April 25 and 26, 1940.

The subject for discussion will be "The Choice of Operation for Glaucoma." The discussion will be opened by Prof. A. J. Ballantyne.

Members wishing to read papers are asked to send their titles to Mr. J. H. Doggart, 49 Wimpole Street, London, W. I., as soon as possible. Abstracts of these papers should be sent not later than March 23, 1940, to be printed for circulation at the congress.

GENERAL

A World Assessment of Blindness.—"The International Association for the Prevention of Blindness undertook in the past years an inquiry into the incidence of blindness throughout the many countries of the world, and of the regulations in force in those countries for the prevention of blindness. This report has now been published in French (*Internat. Ophth. Cong.* [*Concilium ophth.*, 1937] **7**: 1-310, 1938). It is an important piece of work of 310 pages, and full of information collected by the International Association. No fewer than thirty-seven countries responded to the inquiry. It is of much interest to compare the reports and to note the differences shown in them, not only in the variation in the main causes of blindness but also in the number of the blind in relation to the populations. In the East the figures are estimates, and they are high owing mostly to the widespread incidence of trachoma. In some of the Western countries the figures seem to be contradictory, for there is recorded a higher proportion of blindness in some of those countries where we know most is done, and done successfully, for the prevention of blindness. The reason for that apparent contradiction is that in these countries, like England, blindness is measured by its economic disability and not by a total absence of sight. The report is a valuable document and the Egyptian publication is well done."

Correspondence

IS THE ONSET OF INTERSTITIAL KERATITIS RELATED TO RIBOFLAVIN DEFICIENCY?

To the Editor:—A year ago there appeared, almost simultaneously, an article by Bessey and Wolbach (*J. Exper. Med.* **69**: 1 [Jan.] 1939) and one by us (ARCH. OPHTH. **21**: 315 [Feb.] 1939) relating to observations on the cornea of rats fed diets deficient in riboflavin. The vascularization observed was depicted by each of us, the article by Bessey and Wolbach containing some excellent drawings, and our article, a photograph of a cornea the vessels of which had been injected with india ink. Bessey and Wolbach stated: "Vascularization of the cornea is an early and constant phenomenon in albino rats in riboflavin deficiency. It precedes all other demonstrable lesions of the deficiency." We stated: "From our experience with the diet deficient in riboflavin, the most consistent ocular change was the appearance of corneal vascularization. . . . After riboflavin was added, the most uniform effect was the remission of vascularization."

On Dec. 6, 1938, one of us (L. V. J.) discussed with Dr. Arnold Knapp the results of some six months' experience with riboflavin as used for the relief of human vascularization of the cornea, observations having been made on some 40 patients with all sorts of vascularization, from pannus of trachoma to interstitial keratitis. By that time we had observed that rosacea keratitis responded most satisfactorily and interstitial keratitis and the pannus of trachoma most slowly. These studies were made with natural riboflavin, crystallized from milk whey (supplied by general biochemicals division of the S. M. A. Corporation), a small quantity of which was supplied to Dr. Knapp for his clinical use, limited only because of the great expense of its preparation. At this time we considered the minimal daily therapeutic amount to be 3 mg. This is the approximate riboflavin equivalent of 3 quarts (2.8 liters) of milk. Since at that time (and we believe this is still true at the present) commercial vitamin products contained around 50 micrograms of riboflavin, or the equivalent of a twentieth of a quart of milk, per unit of therapy, we felt it wise not to announce our satisfaction with riboflavin, knowing that the result would be a series of disappointing clinical trials made with advertised vitamin products which were almost void of the riboflavin, even though they contained adequate amounts of vitamins A, B_1, D or C.

Ophthalmologists will be disappointed with clinical results if, after reading the accounts widely distributed through the newspapers of all cities, they attempt to give relief to their patients by the use of preparations containing the riboflavin equivalent of a twentieth of a quart of milk! They will be especially disappointed if they take literally the paragraph as it appeared in a copyrighted article by Science Service, as it appeared in the *Cleveland Press* of Feb. 3, 1940 (reported to be

released by Drs. H. D. Kruse, V. P. Sydenstricker, W. H. Sebrell and H. M. Cleckley and announced through the United States Public Health Service).

It was stated: "Babies born with syphilis, formerly thought to be a cause of the eye disease, can now have their eyes cured." These conclusions, the article reports, are based on the observations of 2 cases! Until commercial products contain riboflavin equivalent to at least a glass of milk per unit of therapy, we advise that patients drink milk.

One of us (L. V. J.) has used riboflavin for as long as a year for some patients with active and inactive interstitial keratitis and has observed no such dramatic disappearance of blood vessels. It is his opinion, however, that there is a definite value, since a far greater number of blood vessels than would be ordinarily expected are eventually empty of blood cells and with the slit lamp are seen as empty channels. It appears that from 6 to 9 mg. daily, taken orally, is adequate, and no additional benefits resulted from the intravenous injection of 15 mg. daily.

In cooperation with the department of dermatology of Western Reserve University School of Medicine during the fall of 1938, we attempted to produce interstitial keratitis in rabbits which were infected with syphilis by maintaining them on a diet void of riboflavin, but no vascularization or evidence of inflammation occurred; nor was it possible to secure a litter of rabbits from a doe so infected and maintained on a diet deficient in riboflavin.

It is of chronologic interest only that Dr. Paul György (on whose suggestion the first animal experiments were carried out and who has cooperated as vitamin advisor throughout the complete study) was given permission to discuss our findings concerning vascularization of the human cornea at (1) the Academy of Medicine of Cleveland in a post-graduate lecture delivered on Feb. 13, 1939 (mimeographed copies of which were widely distributed at 10 cents each) and at (2) a meeting of the Milwaukee Pediatric Society in April 1939.

LORAND V. JOHNSON, M.D., Cleveland.
ROBERT E. ECKARDT, M.S., Cleveland.

Obituaries

HARVEY CUSHING, M.D.
1869–1939

HIS CONTRIBUTIONS TO OPHTHALMOLOGY

On April 8, 1869, in Cleveland, Harvey Williams Cushing was born, and on Oct. 7, 1939, in New Haven, Conn., he died, in the seventy-first year of his life. He was the founder of a school of neurosurgery, being one of the few men ever to have founded a distinct school of surgery. His pupils assert that his name will go down in history as that of one of the greatest surgeons of all time. He was a teacher, author, biographer and philosopher. In the six months since his death, eulogies, tributes, appreciations and editorials, in medical and lay papers, have set forth the fruits of his stupendous industry and marvelous technic. Recorded again and again are the customary dates and details. His father and his father's father and his great-grandfather were physicians; so medicine "ran in his blood." Of pure New England stock, transplanted beyond the Alleghanies, it was natural, as so often happens in the unmixed race of New England, that he inherited family traits and the family pursuit. In order to follow him from place to place as he went on his course through life, a few chronologic facts are here recorded. He was graduated from Yale in 1891 with the degree of Bachelor of Arts and from Harvard in 1895 with the degree of Doctor of Medicine and Master of Arts. As to his surgical homes and positions, he was house officer at the Massachusetts General Hospital from 1895 to 1896. The year 1896 marked the beginning of his connection with the Johns Hopkins Hospital and Medical School. One year of this period, which he referred to as the happiest year of his life, he spent in research work abroad, making the trip over the Atlantic with William Osler. He worked under the surgeon Theodor Kocher and the pathologist Hugo Kronecker, both men of great renown, in Berne, Switzerland. He ended the year with the famous pathologist Sir Charles S. Sherrington in Liverpool, England. He had tried to work with Sir Victor Horsley in London, England, but this famous neural surgeon was far too busy to attempt to teach him. Returning to Johns Hopkins, he was promoted from the rank of assistant resident surgeon, and on up until he was made associate in surgery in 1902 and associate professor of surgery in 1903. He occupied these two positions until 1912, when, after sixteen

Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 19, 1940.

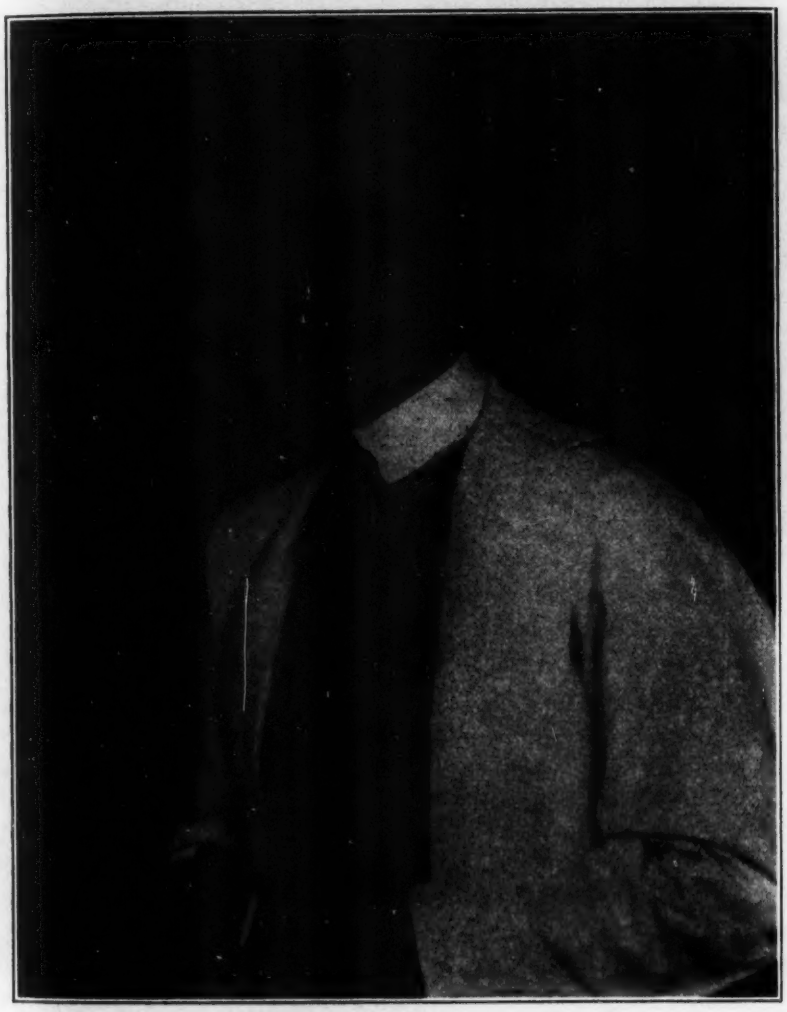
years of service, he returned to his medical alma mater, Harvard, and entered on the duties of surgeon in chief of the newly built Peter Bent Brigham Hospital and professor of surgery in the medical school. In 1932, this being the year set by himself for retirement from active surgical work, he became emeritus in both institutions.

There was an interruption in his Harvard connection from 1917 to 1919, when, with the rank of colonel, he served in the World War as director of the United States Army's Base Hospital No. 5 in France. In 1918 he was appointed senior consultant in neurologic surgery in the American Expeditionary Forces.

In 1933 he was persuaded to return to Yale, from which he had been graduated forty-two years before, to become Sterling professor of neurology. With him went his collection of 2,000 tumors of the brain, with photostatic copies of their histories, and his vast library of medical books, believed of its kind never to have been surpassed by any ever held in private hands. In 1937 he became emeritus. At the time of his death he was director of studies in the history of medicine at Yale. Thus it is seen that his footsteps ended, after forty-eight years, where they began in his preparatory work for medicine. How many times for brief periods was he called away from his surgical homes at Johns Hopkins, Harvard and Yale to deliver lectures and orations or to receive honors and degrees in institutions of learning in this country and abroad!

It was in 1901 that he delivered the Mütter lecture in Philadelphia, at the early age of 32. This lecture was entitled "Some Experimental and Clinical Observations Concerning States of Increased Intracranial Tension." It was based on experimental work that he had done under Kocher in Berne. The lecture was followed two years later, in 1903, by a paper entitled "On Routine Determinations of Arterial Tension in Operating Room and Clinic," read at the Boston Medical Library. This paper marks the beginning of blood pressure determination in this country. Cushing was now launched on his remarkable career as a formal lecturer and deliverer of orations. The topic always fitted the occasion, and many of the papers so presented were of profound scientific importance. His addresses were by no means confined to medical subjects, but, as the event required, he delivered lectures on the history of an institution whose guest he happened to be or on the life and times of some great medical personage. It was said that he always had the "feel" of any institution that he visited and that he made many inquiries about its history and often put to shame those connected with it through knowledge of its traditions greater than theirs. His vacations and peregrinations were different from the standards set by most medical men in that they were given up to lectures or to visits to distant clinics in an insatiable search for knowledge.

In England, in the summer of 1920, unheralded he appeared at the Oxford Ophthalmological Congress. The presence of so distinguished a guest having been made known to the master, he was called on to take part in a discussion on perimetry, to the opening of which he had been listening. He confessed that he had come "to see what an ophthal-



HARVEY CUSHING, M.D.
1869–1939

mologic congress was like" and that he had been most interested in a discussion on the humanities that had preceded the principal subject. He admitted that he had been taking notes and that he had remained longer than he had intended because of his great interest in perimetry as an aid in his neurosurgical work.

In the course of time the figure of this small, alert, wiry man, with a deep mellow voice, became a familiar one in the deliberations of two generations of surgeons, scientists, philosophers, historians and librarians throughout the length and breadth of this country and in Canada and Great Britain.

In addition to the preparation for elaborate lectures and orations and in spite of the precious time consumed in delivering them in distant parts, the heavier work on books and monographs was pursued with a vigor that never abated. The mere enumeration of their titles and dates is as a diary of the days of his years, reflecting the limitless breadth of his activities. In 1912 there came out "Pituitary Body"; in 1917, "Tumors of Nervus Acusticus" and "Story of Base Hospital No. 5"; in 1925, "Life of Sir William Osler"; in 1926, "Classification of Gliomas and Studies in Intracranial Physiology"; in 1927, "Pathological Findings in Acromegaly"; in 1928, "Tumors Arising from Blood Vessels" and "Consecratio medici"; in 1932, "Intracranial Tumors" and "Pituitary Body and Hypothalamus"; in 1936, "From a Surgeon's Journal," and finally, in 1938, "Meningiomas" (with L. Eisenhardt), the largest of his monographs and regarded as his best. What nuggets of purest gold did this master surgeon strew along the pathway of the last six and twenty years of his life, not the least being a description of a syndrome associated with basophilic adenomas of the pituitary gland, now known as "Cushing's disease." Herein lies his noble contribution to endocrinology. Many of his works are replete with references to the eye and are rich in illustrations of the effects of tumors on the visual fields. Every ophthalmologist should have Cushing's volumes on the shelves of his library. They would prove a constant source of information and inspiration.

The sixtieth anniversary of his birth, in 1929, did not pass unheeded. A special number of the *Archives of Surgery* was issued in his honor, containing eighty-two papers. The contributors were former pupils, some of whom by this time had risen to positions of prominence in medicine and surgery. What they had learned from him had come through his example and precept and the good old established method of apprenticeship—"The hippocratic method," as he termed it. Cushing was called "the surgeon of the written word," because from his college days he was in the habit of taking copious notes, embellishing them with accurate and artistic free hand sketches.

The basis of his surgical technic lay in a thorough knowledge of the pure anatomy of the field in which he worked. Experiments, begun in the earliest years of his medical career and never discontinued, contributed to his unrivaled perfection as a neural physiologist. He belonged to the "Halsted school" of surgery, which teaches that deliberation and precision, with perfect hemostasis, lend efficiency and dignity to an

operation. Hitherto, in the preanesthetic days, swiftness was the measure of the ability of a surgeon. This was particularly true of the English surgeon Sir. Astley Cooper, who like Cushing was a prolific writer and a world renowned operator and teacher. But with Cushing, it was far otherwise. His operations on the brain were timed by hours, not minutes. He seldom operated on any other part of the body, although he was surgeon in chief at Harvard. He did not instruct during an operation, but he was a performer, soul and body entering into the task. He lacked the gifts of moving oratory and extemporaneous speaking. Exquisite lightness and dexterity characterized his handling of all tissues. Probably no surgeon ever lived who felt the sanctity of the living normal cell as deeply as he or tried more conscientiously to protect it from unnecessary injury. From all accounts it must have been with him, as his friend John Chalmers Da Costa wrote of Philadelphia's famous surgeon Pancoast, "he had a hand as light as a floating perfume and an eye as quick as a flashing sunbeam."

It is an old aphorism that there is no great reward without great labor. This was true of Cushing. He wrought in hours stolen from family and friends and from periods of much needed rest. He knew what it was to suffer in his own person, for, known to but few, since the World War he often labored under pain due to peripheral vascular disease that came on during his arduous duties in France. Neither was he spared in his surgical life, as he confessed in retrospect "sudden harrowing tragedies which serve to unnerve a conscientious surgeon."

The first signal honor to be conferred on him came in 1913, the year after the publication of "Pituitary Body," in the form of the degree Master of Arts (honorary) from Yale. This same year, as a further indication of the eminence he had attained, he was called to be orator in surgery at the seventeenth International Congress of Medicine at London. During his stay in England he was elected a fellow (honorary) of the Royal College of Surgeons. Degrees, fellowships, titles and honorary memberships followed fast on one another's heels. Particularly flattering was the award of the Pulitzer Prize in Letters of Columbia University in 1926, in recognition of his "Life of Sir William Osler," on which he labored with an amazing amount of collected details for five years. This put the seal of approval on his humanistic accomplishments. Whether in peace or in war, whatever he touched he adorned. Whenever he was asked to go a mile, he went twain! He was made Officer of the Legion of Honor, and he was as worthy of this distinction as Larrey, Napoleon's military surgeon, of whom it is written: "He followed Napoleon with love that never failed, with constancy that never faltered, and gave all his best skill to friend and foe as duty called." From his own country Cushing received the United States Distinguished

Service Medal, and in England he was made Companion of the Bath (military). But the crowning recognition of his merit, in comparison to which all others were as stepping stones, came in 1938, when he crossed the ocean to receive from Oxford University the degree Doctor of Science, "honoris causa," an unprecedented honor for an American physician. To felicitate him on this high occasion, there assembled at Oxford a notable gathering of his former pupils and of colleagues from many countries of Europe. On a Sunday morning he held at Radcliffe Hospital, Oxford, a clinic that those who attended said they would never forget as long as they remembered anything. Only a few weeks before his death there came a cablegram from the Council of the Royal College of Surgeons, London, nominating him for honorary fellowship, to which he cabled back, "Refusal by a surgeon of such an unusual honor inconceivable."

A list of all degrees and honors conferred on him runs into pages. Two there are that concern ophthalmologists particularly. One is the award to Cushing of the Knapp Prize in Ophthalmology in 1928, for the paper entitled "Meningiomas Arising from the Tuberculum Sellae with the Syndrome of Primary Optic Atrophy and Bitemporal Field Defects Combined with a Normal Sella Turcica in a Middle-Aged Person (with L. Eisenhardt)." This, one of Cushing's most informative papers, was read before the Section on Ophthalmology of the American Medical Association at Minneapolis in 1928. The other honor referred to is his election to honorary membership in the American Ophthalmological Society in 1938 in recognition of all that he had done to enlighten, elevate and broaden this special branch of surgery. But if he had helped ophthalmologists, they also had been of help to him. Hear what he modestly said at his seventieth birthday party, given by the Harvey Cushing Society at New Haven, April 8, 1939: "I have spent my neurosurgical life leaning heavily on others: on Percival Bailey and his pupil Louise [Eisenhardt] in histopathology; on Clifford Walker for keeping our perimetry reasonably accurate by periodically coming on from the West Coast to check our procedures; on Larry Reynolds and Merrill Sosman for their unfailing interest in the roentgenological features of the work; on John Fulton for keeping us attuned in these later years to its physiological aspects; and on many more of you for assistance at the operating table, more particularly on Gil Horrax, who for so long a time carried the brunt of the load." Then he referred to the faces that he sorely missed, and he named several as being "doubly dead in that they died so young." Perhaps he had also in mind George Derby, professor of ophthalmology at Harvard, with whom he worked in clinical collaboration and whose memoir he wrote in a touching but searching vein. He could have gone further and declared with Cooper that much of his success depended on his zeal and industry, for

which he could take no credit, as it was given to him from above. Among the spontaneous tributes that he heard at this memorable party were letters from patients bespeaking their inexpressible thankfulness for restoration of their sight. He always asserted that this result was the crux of his operations.

The acknowledgment of his debt to ophthalmologists on his last birthday was not the first. At the meeting of the Oxford Ophthalmological Society, already alluded to, in unpremeditated remarks he referred to Clifford Walker, then on his staff at Harvard, as "a mathematical genius of the Helmholtzian order," who, by refining the technic of perimetry, had made it possible to arrive at a "precocious diagnosis" of very early pressure symptoms, especially of changes in the chiasm due to pressure by pituitary lesions. Cushing and Walker strove to create a standard method for the taking of visual fields, which could be passed on to those interested in ophthalmology "but who in the matter of practice stood only on the threshold of the specialty." To fill this need, Walker devised the special chart and disks and the special technic for the quantitative taking of fields now in use in neurologic clinics. Said Cushing, more important than the instrument is the perimetrist, and he stressed the importance of the peripheral field to the neurosurgeon.

Further elucidation of what came to be known as "Cushing's syndrome" was contained in a paper entitled, "The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adult Patients with a Normal Sella Turcica," read at the thirteenth International Ophthalmologic Congress, at Amsterdam, Netherlands, in 1929. This served to strengthen the link between neurosurgical and ophthalmologic surgeons. The latter were brought to realize the importance of early and accurate perimetry, as patients with tumor of the brain are prone to consult them first.

Cushing more than once referred to the ophthalmologic surgeon as being the oldest of the surgical specialists and to the neurosurgeon as being the youngest. He observed that the neurologists had begun to imitate the ophthalmologists by becoming surgically minded. He spoke of the optic chiasm as constituting "the cross-roads" where ophthalmologists and neurologists have a common place of meeting. He explained that the modern neurosurgeon has come to deal largely with lesions that mechanically affect the intracranial part of the apparatus of vision, whereas the ophthalmologic surgeon limits his operative procedures to the intraorbital portion. He settled the mooted question as to whether meningiomas in the sheath of the optic nerve have an intracranial or an extracranial origin. He proved that they may arise primarily in either location and may progress either way through the optic foramen. He believed in "seeing all round" a tumor. He pointed out that in orbital operations ophthalmologists were in the habit of

attacking a mass from in front or from the side, but he was sure that many of them should be approached to "the windward," that is, from the rear and above, and that only a neurologic surgeon is capable of doing this. He maintained that what is known as "choked disk" is a mechanical rather than an inflammatory process in the vast majority of cases, else vision would not be restored so soon after the mere removal of a tumor. He emphasized that tumors of the brain are too heterogeneous to be grouped together and that they differ not only in type but in sites of predilection and in life history and prognosis. Through his endeavors, that which was once considered hopeless and inoperable in the brain may now be operated on with a degree of security and success to the patient comparable to that given him in an abdominal operation. Happily gone now is the century-old teaching that the cardinal symptoms of a tumor of the brain are headache, vomiting and choked disk.

Admiration, praise and gratitude are written into these lines, which only hint at his achievements. But what was the personal attitude of this man, who was aristocratic and reputed by some to be austere, toward ophthalmologists? The answer may be found in a letter that he wrote to the secretary of the American Ophthalmological Society in 1938, in which, after expressing his deep appreciation and gratitude for the compliment paid him on his election to honorary membership to which he had always aspired, he stated: "My special field of work took me into ophthalmology no less than into neurology, and I have always looked up to the ophthalmologists not only as having been the first surgical specialists but as representing in their persons the elite of the profession."

It is a duty and an honor that this section discharges in pausing to reflect on the life of a peerless surgeon now removed from earthly scenes.

BERNARD SAMUELS.

Obituaries

PATRICK CHALMERS JAMESON, M.D.
1867–1939

Dr. Patrick Chalmers Jameson was born in Kircaldy, Scotland, on Sept. 22, 1867 and died at the Long Island College Hospital on Oct. 27, 1939, after a short illness. He was descended from old Scotch ancestry. His father, Charles Jameson, was a preacher in the Presbyterian church. His mother, Grace Chalmers, was a niece of Dr. Thomas Chalmers, one of Scotland's renowned ministers.

He was the youngest of seven children and obtained his early schooling in Edinburgh, where he also attended the Merchiston Castle School. To recuperate from his strenuous training, and having a great desire to see the world, he came to America and spent four years on various ranches in the Southwest. In Texas he acquired a ranch of his own. Here he developed a strong physique but missed intellectual companionship, and this lack of mental stimulation prompted him to return to Scotland.

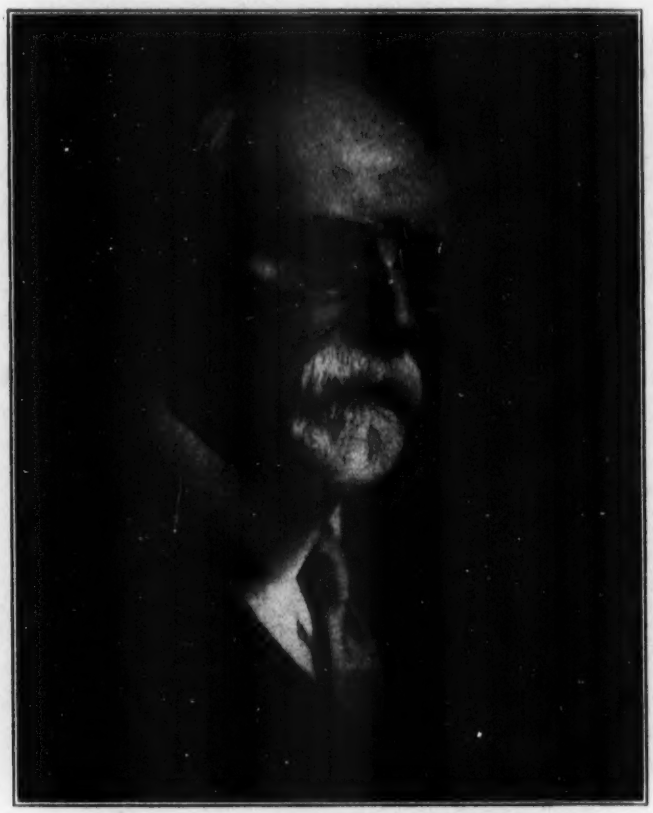
Having set his heart on a profession, he decided to study medicine. He returned to America and entered the Long Island College Hospital in Brooklyn, from which he graduated in 1892. He was influenced in the choice of this school by his great admiration of Prof. Alexander J. C. Skene, who then held the chair of gynecology in that institution. Dr. Skene was also a Scotchman, and the racial ties were strong and always remained so with Dr. Jameson.

He served his internship at the Brooklyn Hospital, and there he became especially interested in surgery and pathologic research, which he continued at the Hoagland Laboratory with Dr. Archibald Murray and the late Dr. Randolph. Later, becoming interested in ophthalmology, he attended the clinics of Dr. John Weeks and the late Dr. Herman Knapp. He also spent several summers in some of the European clinics, especially at Bonn, Utrecht and Wiesbaden with Professor Saemish, Professor Snellen and Dr. Pagenstecher.

His association with the Brooklyn Eye and Ear Hospital began in 1896, under the late Dr. Arthur Mathewson; on the latter's retirement, he took over his private practice. He served the Brooklyn Eye and Ear Hospital for many years: for twenty-six years as a surgeon and for his last seven years in the capacity of senior surgeon. As an active

member of the board of directors and its secretary since 1927, he was most instrumental in obtaining the new hospital building, which was opened in 1930. At the time of his death he was also consulting ophthalmic surgeon to the Brooklyn Hospital, St. Johns Hospital and the Caledonian Hospital.

Dr. Jameson enjoyed a large private practice. His patients were most devoted and loyal to him.



PATRICK CHALMERS JAMESON, M.D.
1867–1939

He was a member of the American Medical Association, the New York state and county medical societies, the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the New York Ophthalmological Society, the Brooklyn Ophthalmological Society, the Associated Physicians of Long Island and the Practitioners Club. He was a fellow of the American College of Surgeons and belonged to the Alumni Society of the Brooklyn Hospital.

Dr. Jameson contributed generously to the various journals, and with a special aptitude toward surgery, he devised and improved several operative procedures. Some of his papers are:

Observations on the Prophylaxis of Ophthalmia Neonatorum, 1899.

Injury of an Eye, Necessitating Enucleation Twenty Years Later, *Brooklyn Medical Journal*, 1900.

The Bacteriological Element in the Etiology of Acute Catarrhal Conjunctivitis, *Brooklyn Medical Journal*, 1902.

Atropia: Its Uses and Contraindications in Ophthalmic Practice, *Brooklyn Medical Journal*, 1903.

Report of Two Cases of Iritis with Comparative Remarks, *Brooklyn Medical Journal*, 1904.

Re-Attachment in Iridodialysis: A Method Which Does Not Incarcerate the Iris, *Archives of Ophthalmology*, 1909.

Replacement of Incarceration of the Iris by Traction from Within the Anterior Chamber, *Archives of Ophthalmology*, 1909.

Surgical Treatment of Wounds of Cornea with Prolapsed Iris, *Archives of Ophthalmology*, 1926.

Schaaf Forceps for Removal of Foreign Bodies, *Archives of Ophthalmology*, 1931.

Surgical Entity of Muscle Recession, *Archives of Ophthalmology*, 1931.

Some Essentials and Securities Which Stabilize Operations on Ocular Muscles, *Archives of Ophthalmology*, 1932.

Vascularization of Anterior Segment of Eye: Bearing of These Studies on Some Operative Procedures, Including Possible Supplementary Procedure for Glaucoma; Preliminary Report, *Archives of Ophthalmology*, 1933.

Use of Thyroxin in Ophthalmology: Its Application as a Local Agent and Its Action as Metabolic Alterative, *Archives of Ophthalmology*, 1934.

Ligated Suture, *Archives of Ophthalmology*, 1934.

Some Observations as to Indications for Advancement and Kindred Operations, *Archives of Ophthalmology*, 1935.

Brooklyn Eye and Ear Hospital, 1868-1935, *Archives of Ophthalmology*, 1935.

Operative Treatment of Detached Retina: Principles Observed by Six Individual Operators, *Archives of Ophthalmology*, 1936.

Recession Operation (Reply to Dr. O'Connor), *American Journal of Ophthalmology*, 1936.

Sub-Conjunctival Section of the Ductules of the Lacrimal Gland as a Cure for Epiphora, *Archives of Ophthalmology*, 1937.

Surgical Management of Ptosis with Special Reference to Use of Superior Rectus Muscle, *Archives of Ophthalmology*, 1937.

Technique of Scleral Fixation of Extra-Ocular Muscles, *American Journal of Surgery*, 1938.

Entity of Muscle Recession: Short Résumé of Its Technique and Principles with New Supplementary Notes and Illustrations, *Archives of Ophthalmology*, 1939.

In 1922 he published his paper on "The Correction of Squint by Muscle Recession with Scleral Suturing." This operation is now familiarly known as the Jameson recession operation.

He was regular in attendance at the meetings of his local and national societies and usually contributed to the discussions.

He was tall and slim, soft spoken and kindly in manner, with a genial and cordial personality which endeared him to his friends and associates. The sincerity and staunchness of his character reflected his firm convictions; from these he was not easily swayed. A senior member of his profession, he was most considerate of the younger practitioners, giving to many a helping hand.

Dr. Jameson was an extensive traveler, spending many summers abroad, especially in the British Isles. On such trips he often visited the foreign hospitals and clinics.

A great lover of nature, he sought the wide open spaces and enjoyed long hikes through the country. With a keen sense of humor, he delighted to tell his Scotch stories, and he told them well.

In 1894 he married Miss Sarah Hanmer and had one daughter, Miss Jeanette Jameson, both of whom survive him.

He was a member of the old Hamilton Club of Brooklyn and attended the Fifth Avenue Presbyterian Church in New York.

Dr. Jameson will be remembered by his many friends, patients and associates as a skilled and conscientious physician and as a high type of gentleman with lofty principles and high ideals.

JOHN H. OHLY.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

STAPHYLOCOCCUS TOXIN. J. H. ALLEN and A. E. BRALEY, Am. J. Ophth. **22:** 11 (Jan.) 1939.

After a study of the effects of staphylococcus toxin on rabbits, Allen and Braley reach these conclusions:

"1. The pathogenic action of ocular strains of staphylotoxin is similar to that of general strains, with perhaps the exception of the action upon the gastrointestinal tract.

"2. The hemolytic unit apparently is satisfactory for estimating the potency of ocular staphylotoxin.

"3. Serologic studies should be made upon all rabbits used for experimental study with staphylococci and their toxins."

W. S. REESE.

Biochemistry

TESTS IN CHEMICAL AND PHYSICOCHEMICAL TOPOGRAPHY OF THE LENS. E. TRON, Vestnik oftal. **14:** 59, 1939.

This article is extensive and lends itself to abstracting with difficulty. Tron devoted this part of the work (the second part will follow) to the study of the physicochemical topography of the lens, by comparing its anterior and posterior cortical layers and their reaction with similar external factors. "Salt" cataract obtained by immersion of the lens of the bull's eye in hypertonic Ringer-Lock solution served as an object for the study. The difference in the usual technic was that a portion of the sclera and ciliary body was left attached to the lens; a glass curved rod was put through this portion, so that the lens was hanging freely in the solution, and both its surfaces could be seen and examined with the slit lamp.

Different changes were observed on the two surfaces of the lens both in the early and in the late stage. In the early stage of cataract development (caused chiefly by the loss of water by the lens) a large number of large vacuoles were found on the anterior surface of the lens, while on the posterior surface only a few small vacuoles formed, which would soon disappear. The later stage, characterized by the formation of dense chalklike opacities of the superficial layers of the lens with their consequent degeneration, different intensity and different time of development of the process on both surfaces, was observed.

In salt cataract due to immersion of the lens in a copper sulfate solution and mainly to precipitation of the lens proteins, there were also observed different changes on the two surfaces, both in the time of formation of the opacities and in their morphologic structure.

The tests with partial removal of the lens capsule demonstrated first that in the absence of the capsule the opacities varied on the anterior

and posterior surfaces, and secondly, that the opacities varied on the same surface according to the presence or absence of the capsule.

The work is to be continued.

O. SITCHEVSKA.

Comparative Ophthalmology

THE BINOCULAR VISUAL SPACE OF DOMESTIC ANIMALS. A. PISA, Arch. f. Ophth. **140**: 1 (Feb.) 1939.

In 1826 Johannes Müller, by rather crude experiments, established the fact that animals with laterally directed eyes have a binocular visual space of considerable size, which fact accounted for the obviously good depth perception of some of those animals, especially birds. In 1902 Tschermak-Seysenegg made measurements of the horizontal expanse of this binocular visual space of various domestic animals. The whole head of the freshly killed animal was frozen, the orbital roofs were removed by careful dissection and the outlines of the binocular field were determined by watching through the sclera the retinal images of a point of light located in front of the animal's head. Perimetric study was thus carried out by direct objective observation of the retinal images. The rabbit, for instance, was found to have a binocular visual space measuring about 34 degrees in its horizontal diameter. Tschermak-Seysenegg concluded that in the visual space of most vertebrates, including animals with total decussation of the optic nerve fibers in the chiasm, there is at least a small area within which sensory cooperation of the two retinas is possible. Thus Tschermak-Seysenegg proved the incorrectness of the concept of a direct proportionality between the number of uncrossed fibers in the chiasm and the extent of the binocular visual space.

Pisa has continued the work of Tschermak-Seysenegg (under the latter's supervision) and has concerned himself chiefly with the question to what extent and in which direction the visual axes of freshly killed domestic animals deviate from their in vivo position. Thereby he arrives, with complicated methods, at figures which probably apply to the practical binocular visual space.

P. C. KRONFELD.

Cornea and Sclera

SUPERFICIAL MARGINAL KERATITIS. O. H. ELLIS, Am. J. Ophth. **22**: 161 (Feb.) 1939.

Ellis briefly discusses marginal ulcers of the cornea and superficial marginal keratitis in particular. He reports 2 cases and gives the following summary:

"The first case of superficial marginal keratitis herein described presented severe bilateral involvement, which greatly interfered with vision and caused intense pain, necessitating the enucleation of both eyes. Opportunity was thus offered for describing the anatomical findings in this condition. This is the first time that the anatomical findings of superficial marginal keratitis have been published. A second case with the clinical findings of superficial marginal keratitis is reported."

W. S. REESE.

TREATMENT OF KERATITIS WITH HYPOPYON BY THERMOPUNCTURE OF THE CORNEA. G. PACALIN, Bull. Soc. d'opht. de Paris **50:** 42 (Jan.) 1939.

Pacalin claims to have been the first to replace the Saemisch section by puncture of the cornea with galvanocautery (*Arch. d'opht.* **48:** 498 [July] 1931). Puncture relieves the increased pressure in the aqueous, helps in nutrition of the cornea and allows for additional immune substance to be accumulated in the secondary aqueous. The same type of cautery is used as that which the author recommends for retinal detachment. Two case histories are given to show the efficacy of the procedure. The relief of the pain is immediate. If there is any reason to believe that there is infection in the lacrimal tract, the puncta are sealed by galvanocautery. Neither the use of antiseptics nor further surgical procedures are necessary. L. L. MAYER.

MEGALOCORNEA IN CONNECTION WITH OTHER PHYSICAL IRREGULARITIES IN MEMBERS OF THE SAME FAMILY: REPORT OF CASES. R. G. POSTHUMUS, Klin. Monatsbl. f. Augenh. **102:** 1 (Jan.) 1939.

Posthumus reports 3 cases of megalocornea in a family two previous generations of which had been free from this disease. He found diabetes, labyrinthine deafness and nephritis in several members of this family; the hair was poorly pigmented, especially in the men. Extraction of a nuclear cataract in 1 adult is described, vision of 8/10 having been obtained. In none of the patients with megalocornea was the globe enlarged; atrophy of the vitreous and degeneration of the pigment of the iris occurred. Typical receding of the forehead was noted in some of these patients, but this symptom cannot be regarded with certainty as hereditary. Slight mental debility of these patients cannot be evaluated reliably from a psychic or physical standpoint.

K. L. STOLL.

ULCUS RODENS MOOREN. E. DALSGAARD-NIELSEN, Acta ophth. **17:** 28, 1939.

This article contains clinical reports of 2 cases of Mooren's ulcer. One eye was enucleated, and the histologic changes are described. These consisted of thinning of the affected portion of the cornea, proliferation of epithelium, destruction of Bowman's membrane and preservation of Descemet's membrane, newly formed vessels and islands of migratory cells. These pathologic changes occurred in portions of the cornea which macroscopically seemed healthy. O. P. PERKINS.

Experimental Pathology

HISTOLOGICAL STUDY OF THE EYES OF RABBITS CONGENITALLY INFECTED WITH SYPHILIS. H. SHIGA, Am. J. Ophth. **22:** 119 (Feb.) 1939.

On the basis of an experimental study of the eyes of rabbits congenitally infected with syphilis, Shiga makes the following conclusions:

"One hundred forty-one eyes of 71 rabbits with congenital syphilis, ranging in age from very young fetuses to 93 days, were examined.

Thirty-four eyes of 17 normal rabbits of corresponding ages were taken as controls. The conclusions are as follows:

"1. Congenitally syphilitic rabbits are very liable to be born prematurely by abortion. Even if born at full term, they often die soon after birth. Their general development is below normal, and the development of ocular tissue is deficient as compared with that of normal rabbits.

"2. Pathological changes noted are as follows: (a) In the premature fetus and dead fetus born at full term—edema in the subcutaneous tissue of both lids; hyperemia and hemorrhage in the subcutaneous tissue of both lids, extrinsic ocular muscles, in the neighborhood of the lacrimal gland, conjunctiva, subconjunctival tissue, and the cornea.

"(b) In the young which died shortly after birth—infiltration of round cells in the limbus, the sclerotic, the iris, the ciliary body, and extrinsic ocular muscles.

"(c) In two young (aged 2 months) with corneal ulcer—in one of these I found spirochaetae in the cornea. Also prominent cell infiltration (pseudo-eosinophilic leucocytes) could be seen, especially in the anterior layer of the substantia propria, accompanied by a wide area of epithelial defect. The limbus (remote from the ulcer) showed such syphilitic changes as perivascular cell infiltration, vascularization directly under the corneal epithelium near the limbus, and also in the inner layer of the limbus, with irregular curling of fibrous tissue, thickening of capillary vessel walls, and other changes.

"Epithelial defect with subepithelial change is suggestive of a few cases of keratitis of the human fetus (congenital syphilis cases, or those supposed to be such) reported by H. v. Hippel, Reis, Cattaneo, and Seefelder, and my case is identified as a special type of interstitial keratitis, occurring in congenital syphilis of the rabbit, somewhat different from that in acquired syphilis.

"3. Spirochaetae pallidae were found in the cornea and the sclerotic, sometimes accompanied by pathologic changes, sometimes not."

W. S. Reese.

The Metabolism of the Retina After Interruption of the Retinal Circulation. A. Santoni, Ann. di ottal. e clin. ocul. 67: 299 (April) 1939.

The author placed a ligature about the entire orbital contents of rabbits and rats, controlling the changes in the fundus with the ophthalmoscope to be sure that obliteration of the retinal circulation was complete. The ligature was left in place for fifteen to ninety minutes, and the eyes were removed after ten to twelve days and the retina placed in the Warburg respirator, the retina of the fellow eye being used as the control. The oxidative power of the retina showed an increase after interruption of the circulation for fifteen to thirty minutes, while after the ligature was retained for sixty to ninety minutes a decrease in oxidation was noted, averaging 25 per cent of the normal. Santoni believes that the degenerative changes resulting from shorter interruptions of

the circulation free autolytic ferments and fatty acids, which increase oxidation, while after degeneration has reached a certain degree this increased activity no longer occurs and oxidation drops below normal.

S. R. GIFFORD.

General Diseases

BILATERAL RETROBULBAR NEURITIS AS A BEGINNING SIGN OF PERNICIOUS ANEMIA. L. PAUFIQUE, Bull. Soc. d'opht. de Paris **50:** 144 (Feb.) 1939.

It is not unusual to find retrobulbar neuritis in the advanced stage of pernicious anemia, but visual difficulties are unusual in the early stages. A man of 45 complained of gradual loss of vision for the past thirty days. Visual acuity was 3/10 in the right eye and 3/10 in the left eye. The refraction and pupils were normal, as were the peripheral fields. However, there were bilateral central scotomas for red and green. There was a subicteric tinge. The teeth were badly infected. The patient drank 2 liters of wine a day and was an excessive smoker. A diagnosis of toxic amblyopia was made. In spite of care of the teeth and limitation of smoking and drinking, the condition became rapidly worse. Because of the pallor of the skin and mucous membranes, a complete examination of the blood was ordered. There were 2,000,000 red cells and a hemoglobin content of 42 per cent with an index of 1. There were marked anisocytosis and poikilocytosis. Under treatment, recovery was prompt. In one month the visual acuity had returned to 0.8, and the central scotomas had disappeared.

L. L. MAYER.

Hygiene, Sociology, Education and History

GLASSES FOR PREVENTION OF GLARE IN NIGHT DRIVING. H. VIALLEFONT, Bull. Soc. d'opht. de Paris **50:** 97 (Feb.) 1939.

Grave accidents may occur because of the glare of the lights from an oncoming automobile. An ideal protection consists of a lens which utilizes all vision in darkness but shuts out glare when it appears. This may readily be arranged by gluing a small segment of neutral glass at the upper right corner of each lens. This segment interferes in no way with direct vision, and only when the head is tilted will the protective glass come into usage. A photograph of the glasses is shown.

L. L. MAYER.

Methods of Examination

A PERFECTION SCOTOMETER. F. A. JULER, Brit. J. Ophth. **23:** 239 (April) 1939.

The apparatus described by Juler aims at the same results as the Bishop Harman instrument, the test object being a spot of light on a gray surface. The advantage claimed for the instrument is that it is less complicated than the one devised by Harman.

The article is illustrated.

W. ZENTMAYER.

UTILITY OF ANGIOSCOTOMETRIC EXAMINATION IN THE DIAGNOSIS OF EDEMA OF THE PAPILLA. A. MAGITOT and A. DUBOIS-POULSEN, Bull. Soc. d'opht. de Paris **50:** 32 (Jan.) 1939.

Angioscotometric examination has been used with much success in the study of the vascular tree of the retina. With a 0.5 mm. test object, deviation in the extent of Mariotte's blindspot are well delineated. In this article the authors report on a study of pathologic blindspots of Mariotte. Factors influencing the size were found to be voluntary apnea, cervical constriction, previous use of the electric light of the ophthalmoscope and pressure on the globe.

In cases of hyperopia with pseudoneuritis, the blindspots were found to be normal by angioscotometric examination. If any enlargement occurred in such cases, search for the cause of hyperemia of the disk was sought. Frequently, as demonstrated by Evans, hyperemia of the disk with enlargement on angioscotometric examination has been found to be due to involvement of a sinus.

In endocrine disorders and even at menstruation, edema of the nerve head occurs with enlargement of the blindspot. In stasus and papillitis, minute changes in the outline of the disk are readily found with angioscotometric examination, and it is relatively simple to watch the progress of an increased intracranial pressure by this method.

As a matter of fact, the authors believe that this form of examination is the only one which distinguishes between stasus and papillitis in many cases. One is better able to distinguish the absolute central blind area and the peripheral amblyopic zone by his method. The authors cannot decide whether Evans' hypothesis is justified, in which he attributes the amblyopic zone to the presence of a large number of small vessels which arch over the disk.

L. L. MAYER.

Neurology

FACIAL HEMIATROPHY. F. B. WALSH, Am. J. Ophth. **22:** 1 (Jan.) 1939.

Walsh reports 2 cases of facial hemiatrophy illustrating the two types, congenital, or nonprogressive, and the more frequent progressive type. He reviews the theories explaining this condition and discusses its relation and resemblance to scleroderma. The second case reported was unique in that heart block was present. Walsh enumerates the ocular signs that occur, though these are not constant.

W. S. REESE.

OCULAR SYMPTOMS IN SCHIZOPHRENIC PATIENTS TREATED WITH METRAZOL (CARDIAZOL) AND WITH INSULIN SHOCK THERAPY. V. CAVKA, Arch. f. Psychiat. **109:** 721 (April) 1939.

Ten schizophrenic patients who were treated with metrazol (cardiazol) and 20 who received insulin shock therapy were studied for ocular changes before, during and after the convulsion. Of the first group, no abnormalities of the fundi were evident before treatment.

Six were emmetropic, 4 were hyperopic and 1 was myopic with conus. Cavka demonstrates his observations on this group in a table. The outstanding characteristic was blepharospasm with conjugate deviation to the right and upward during the reaction. The pupils were widely dilated. The retinal arteries were reduced to one-fourth their caliber, and the veins were dilated, so that the relation before the reaction was 1 : 2.5 instead of 1 : 1.5.

In 2 patients there was a blurring of the outlines of the disks, which disappeared several days after the reaction. No hemorrhages were noted. Cavka feels that such symptoms are comparable to the usual attack and changes in the fundus in idiopathic epilepsy.

The group given insulin therapy showed similar changes. The intraocular tension of 13 persons in this group was raised.

L. L. MAYER.

PERMANENT LESION OF THE VISUAL TRACT DUE TO FLYING AT HIGH ALTITUDES: REPORT OF A CASE. W. LÖHLEIN, Klin. Monatsbl. f. Augenh. **101:** 818 (Dec.) 1938.

The patient, aged 34, who had been an aviator for several years, had escaped death in an aerial accident in 1937 in which his companion died. A few weeks later, ascending to an altitude of from 5,000 to 6,000 meters, he suddenly experienced severe headache, vertigo and obscuration of the field of vision. He was barely able to land his aeroplane. His companion showed no symptoms except severe epistaxis, while the patient himself, after landing, presented vertigo, nystagmus, Romberg's symptom, right hemianesthesia and right homonymous hemianopia due to total scotomas in the central zone of each eye. Few neurologic symptoms were noted. The ocular symptoms were unchanged after a period of rest of three months. Vision in each eye remained normal. Löhlein interprets the symptoms as the result of a lesion of the left cerebral hemisphere, possibly toward the left inner capsule. The sudden onset, the absence of an increase of the symptoms, the fairly negative roentgenographic findings and the negative result of the test of the spinal fluid are indicative, in the author's mind, of a vascular disturbance in the left cerebrum, which may have been a vascular spasm or a hemorrhage. Lack of oxygen and fast ascendance may be the source of the symptoms in both men. The graver disturbance in the patient described may be attributed to individual susceptibility or perhaps to some kind of a mechanical lesion of the blood vessels as a result of the accident suffered four months previously.

K. L. STOLL.

EXOPHTHALMOS COINCIDENT WITH INTRACRANIAL TUMORS. H. SKYDSGAARD, Acta ophth. **16:** 474, 1938.

The author limits his remarks to tumors arising from the cerebrum, meninges and cerebral vessels. Exophthalmos was found in 14 of a series of 352 patients with such tumors, seen at the Rigshospital. The exophthalmos was unilateral in 9.

The tumors are tabulated as follows:

Meningioma of the pterygoid bone	5	(unilateral 4) (bilateral 1)
Parasagittal meningioma	1	(bilateral)
Convexity meningioma	1	(unilateral)
Meningioma of the olfactory nerve	1	(bilateral)
Neurinoma of the acoustic nerve	1	(unilateral)
Tumor of the hypophysis	3	(unilateral)
Aneurysm of the carotid artery	1	(bilateral)
Glioma of the temporal lobe	1	(bilateral)

Similar sets of figures from the published series of other authors are given.

The author remarks that while papilledema and exophthalmos may occur together, they by no means necessarily do so; nor does exophthalmos seem to have any definite relation to increased intracranial pressure.

O. P. PERKINS.

CHANGES IN THE FUNDUS OCULI AND PERSISTING INJURIES TO THE EYE IN MIGRAINE. H. GRONVALL, Acta ophth. **16:** 602, 1938.

An 18 year old girl who had suffered from typical attacks of migraine for six years suddenly became blind in the right eye after a prodromal period of giddiness and scintillations. Ten minutes later the lower portion of the visual field was restored, a defect above remaining. Ophthalmoscopic examination twenty-four hours later revealed an area of retinal edema and obliteration of the inferior temporal branch of the central retinal artery. The edema gradually subsided, but the arterial change persisted during the two months that the patient was observed.

No source of an embolus could be found, and the case was regarded as one of arterial spasm associated with migraine.

O. P. PERKINS.

Ocular Muscles

CONTRIBUTION TO THE STUDY OF ACQUIRED SPASMS OF THE LEVATOR MUSCLE OF THE UPPER LID. G. E. JAYLE, Ann. d'ocul. **176:**1 (Jan.) 1939.

In spite of numerous researches on acquired spasms of the levator muscle of the upper lid, the particular characters of this condition as well as the symptoms are not well understood. The question of the pathogenesis also remains in dispute.

Its position among other retractions of the upper lid is not precise, some investigators placing it with the palpable retractions. Certain authors claim that the palpable retractions of the striated type and the retractions of the sympathetic type are attributable to identical lesions.

The main division of the article deals with binocular spasms of the levator muscles of the upper lid, which material is subdivided into that dealing with permanent and intermittent retractions; the symptoms associated with former, such as oculomotor disturbances, Parinaud's syndrome, peripheral paralysis, pupillary changes, exophthalmos, vestibular disturbances, oculocephalic symptoms and general disturbances, and pathogenesis.

The intermittent retractions are discussed under synkinetic spasms, effort spasms and fixation spasms.

The article is to be continued.

S. H. MCKEE.

Pharmacology

PONTOCAINE, THE CAUSE OF PROFESSIONAL ECZEMA AMONG OCULISTS. N. SHIMKIN, Ann. d'ocul. **176:** 198 (March) 1939.

Pontocaine is an anesthetic produced to replace cocaine. It is a derivative of procaine, plus a radical "butyl," the ethyl group being here replaced by the methyl group.

The author reports a case in which palpable eczema developed after the instillation of 1 to 2 drops of a 0.5 to 1 per cent solution of pontocaine hydrochloride. The condition developed within two to three hours and was manifest by a burning sensation in the eyes with redness and edema of the skin of the lids. After six to eight hours a typical eczema of the lids appeared. This condition lasts from eight to fourteen days but disappears completely with treatment by cold compresses. A number of cases of eczema of the fingers, particularly of the nails, have been observed in oculists after the use of pontocaine.

The author himself suffered from eczema of the hands and of the left leg during four years' use of the drug in his clinic. The condition was completely corrected when pontocaine was no longer employed. Other instances are cited similar to the author's own experience.

S. H. McKEE.

ACTION OF ATROPINE ON THE ENUCLEATED EYE OF THE FROG. J. REGNIER and A. QUEAUVILLER, Compt. rend. Soc. de biol. **130:** 1212, 1215 and 1461, 1939.

The enucleated frog's eye kept in mild diffuse light regularly becomes miotic, both in Ringer's solution and in isotonic dextrose solution, reaching its limit by the second hour. After one hour the pupillary area is generally reduced by 30 per cent. The relative action of atropine on the enucleated eye is most marked in the first hour and is greater in Ringer's solution than in isotonic dextrose solution. In the same medium and at the same p_H the mydriatic action of the sulfate is twice that of the citrate or the phenylpropionate salt. Since none of the negative ions (chloride, citrate, sulfate or phenylpropionate) have any topical effect on the pupil, the specific properties of the anion hence do not explain the differences in action of the various salts of atropine.

J. E. LEBENSOHN.

The Pupil

ARGYLL ROBERTSON PUPILS IN ALCOHOLISM. M. HERMAN, Arch. Neurol. & Psychiat. **41:** 800 (April) 1939.

The author reports 3 cases illustrating the association of an Argyll Robertson pupil with chronic alcoholism. The summary follows:

"In case 1 there was evidence of widespread damage to the central nervous system, as shown by the Korsakoff type of mental change, the peripheral neuritis and the pellagrous lesion. In case 2 the classic pupillary change was found, together with extensive peripheral neuritis in the legs. In case 3 there was a transitory pupillary reaction of the Argyll Robertson type during the course of delirium tremens. Wherever the pathologic lesion that is responsible for an Argyll Robertson pupil

exists in cases of neurosyphilis, it seems that a similar pupillary change may be produced in other conditions. In cases of chronic alcoholism, the Argyll Robertson pupil was found accompanying other neurologic disturbances. It is interesting to note that this manifestation may be transitory, as in case 3. Possibly, with repeated attacks a permanent pupillary change may result."

R. IRVINE.

Physiology

THE CHEMICAL EQUILIBRIUM OF THE INTERSTITIAL FLUIDS AND THE AQUEOUS HUMOUR. J. D. ROBERTSON, Brit. J. Ophth. **23**: 170 (March) 1939.

This article is limited to the discussion of the chemical equilibrium that exists (1) between blood and lymph, (2) between blood and gastric juice and (3) between blood and aqueous humor.

The following summary is given by the author: "I have already stated, glucose, urea, and creatinine are freely diffusible and entirely filtrable both in vivo and vitro so that if the membrane in the eye and the choroid plexus allowed simple dialysis to occur, these substances should occur in concentrations equal to that in the plasma; but they do not. It would appear in fact that glucose, urea, and creatinine, diffuse through the stomach wall with no less difficulty than they diffuse into the aqueous humour. In both the stomach and the eye, there is a marked deficiency in the urea, sugar, and creatinine concentrations when these are compared with the concentration of these substances in the blood. In the case of creatinine, all the experiments showed that this substance diffused into the stomach much more readily than into the aqueous humour. It has been stated that it is a second membrane re-inforcing the capillaries of the ciliary processes which is responsible for the low protein content of the aqueous humour, and the impaired filtration of freely diffusible substances such as urea, glucose, creatinine, and uric acid. This so-called reinforced membrane behaves not unlike the secreting membrane present in the stomach, for it causes a similar concentration gradient for diffusible substances.

"When all these facts I have mentioned are considered, it becomes clear that we cannot consider the aqueous humor a simple, protein-free ultrafiltrate or dialysate of blood plasma, but that it is a highly specialised fluid manufactured for a specific purpose. As we found the simple laws governing the formation and absorption of tissue fluid inapplicable to the production of the aqueous humour, so we find the chemical analysis too complex to be explained by the simple laws governing the chemical equilibrium of dialysates." W. ZENTMAYER.

Retina and Optic Nerve

SOLITARY RETINOCHOROIDITIS WITH REFERENCE TO RETINOCHOROIDITIS JUXTAPAPILLARIS-JENSEN. C. HEATH, Brit. J. Ophth. **23**: 289 (May) 1939.

Heath prefers the term solitary retinochoroiditis for the disease to which Jensen's name is usually given. Attention is called to the fact that six years prior to Jensen's paper H. Friedenwald gave a far more

comprehensive description of similar cases and mentioned that the condition had already been recognized by Griffith. It is proper to state, however, that Jensen restricted the application of the term retinochoroiditis juxtapapillaris to those cases in which the lesion is in contact with the disk and produces a sector-like defect in the field of vision. Heath describes a typical case of disease as follows:

An apparently healthy young adult complains that the sight of one eye has been blurred for two or three days. The vision in that eye is found to be reduced to about 6/12. External examination shows nothing amiss. Examination of the fundus shows a vitreous haze which obscures the finer details, but close to the disk is seen a patch of shining, cream-colored exudate shading off gradually into the normal retina on all sides. A week or more later the vision in that eye has deteriorated to about 6/36. Fine keratic precipitates are present, and the vitreous haze is thicker than before. The exudate and the rest of the fundus appear unchanged. The condition remains the same for one or two weeks and then steadily improves. In three months the media are clear, and vision is restored to normal. The fundus shows a pigmented scar corresponding to the site of the exudate, but it is otherwise normal. The field of vision shows an absolute sector defect extending from the blind spot to the periphery.

Twenty-four cases were investigated and the findings compared with those recorded in the literature. An attempt has been made to define criteria for diagnosis. Points of particular interest which emerge are:

"1. The apparent high incidence of the disease in childhood.

"2. The frequent presence of veils in the vitreous.

"3. One case in which the development of anomalous retinal vessels was preceded by retinal ischaemia.

"4. The defects in the field of vision may be wholly or partly relative and may diminish with healing.

"5. One case in which a defect in the visual field was relative throughout after the first attack but became absolute after the second.

"6. One case in which two separate sector-defects in the visual field were produced by two apparently confluent scars.

"7. Defects originating in the nasal visual field appear to be incompatible with the conventional conception of the distribution of the nerve-fibres of the retina."

Heath gives a detailed discussion of the subjective and objective symptoms of the disease as found in his own case reports and those in the literature.

The article is illustrated. W. ZENTMAYER.

ENDOCRINOLOGIC REPORTS ON RETINITIS PIGMENTOSA. E. BIRÓ, Ann. d'ocul. **176:** 293 (April) 1939.

It is only within the last ten years that pigmentary degeneration of the retina has been investigated from an endocrinologic point of view. Up to this date most research has been along anatomicopathologic lines, a satisfactory explanation of the cause and origin of the pigmented retina being sought in histologic study. Investigators of the last thirty

years have searched for, and are still seeking to find, evidence to show whether the displaced pigment is of choroidal or of retinal origin and whether the first changes in the disease are in the choriocapillary layer of the choroid or in the neuroepithelial layer of the retina.

The author's ideas on the causation of retinitis pigmentosa have been influenced by the finding of a relation between the endocrine glands and pigmentary retinitis. Experimental research is being continued with extracts of ovary and testis and with liver extracts. These experiments and their results are to be published later.

S. H. McKee.

Trachoma

Bee Venom in the Treatment of Trachoma. A. Lagomarsino, Ann. di ottal. e clin. ocul. **67**: 143 (Feb.) 1939.

The preparation of bee venom, as described by Brecher, and the literature on its use in trachoma are reviewed. Twenty patients with trachoma in various stages were given subconjunctival injections of the venom mixed just before injection with a local anesthetic. Half an ampule was injected in the upper fold and the other half into the tarsal conjunctiva and tarsus. In cases in which there were pannus and corneal lesions, half an ampule was injected under the bulbar conjunctiva near the limbus. Six injections were usually given at intervals, depending on the duration of the reactions. Treatments were not painful and were well tolerated. The effects were favorable, especially on the corneal lesions, which cleared rapidly. The vessels in some cases disappeared, and corneas that were affected by thin opacities became much clearer. Corneal ulcers healed under treatment, with minimal scarring.

S. R. Gifford.

Society Transactions

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Forty-Fourth Annual Meeting, Oct. 8-13, 1939

Essential Hypertension from the Standpoint of Otolaryngology.[1]
DR. A. C. FURSTENBERG, DR. J. M. MAXWELL (by invitation) and DR. G. H. ALEXANDER (by invitation), Ann Arbor Mich.

While the cause of essential hypertension is not yet clearly understood, a distinct hereditary tendency is recognized, as shown by the numerous reports of cardiovascular symptoms or accidents which appear in the family histories.

From a practical standpoint, it is of utmost importance that the otolaryngologist recognize the occurrence of varying degrees of hypertension and the fact that high blood pressure is not a constant finding in the early stages of the disease. Only repeated examinations may reveal the occurrence and extent of the disease, as the patient frequently consults the otolaryngologist on account of headache. This symptom dominated the clinical picture in 50 per cent of 156 cases of essential hypertension that were extensively studied at the University of Michigan Hospital. Many of the patients reported a previous diagnosis of sinus disease, but only 12 per cent had pain that was frontal in character and of the pressure type that might be confused with the pain of sinusitis. The headache was frequently nocturnal, dull and boring, reaching its maximum intensity in the early morning and vanishing completely in the afternoon. Invariably the patient stated that the headaches were aggravated by emotional excitement, nervousness and fatigue.

Another common symptom of hypertension and, next to headache, the most annoying disturbance of the disease is vertigo. It was presented by 30 per cent of the patients. It is usually described as a feeling of unsteadiness or a "swimming of the head" and is often initiated by a sudden change of position or a quick motion of the head; it is ordinarily relieved by a short period of rest.

It was found that nearly all of the patients presented emotional disturbances or periods of irritability, anxiety and apprehension. One of the

1. This paper and the following two on essential hypertension were presented as a symposium on this subject at a joint session of the Section on Ophthalmology and the Section on Otolaryngology.

most annoying and occasionally terrifying experiences is epistaxis. This symptom occurred in 17 per cent of the cases. The bleeding comes from the septal branch of the sphenopalatine artery, where it anastomoses with a branch of the descending palatine artery on the anterior portion of the quadrangular cartilage.

It is an astonishing fact that patients with hypertension tolerate surgical intervention admirably well. Care should be taken to avoid unnecessary traumatism of tissues and careful attention should be given to ligation of blood vessels of large enough caliber to favor hemorrhage. If a general anesthetic is required, ether, perhaps, administered by the open or intratracheal technic, is the safest. Danger from the anesthetic is minimized if the blood pressure can be maintained at a nearly constant level during operation. To do this it occasionally becomes necessary to administer fluids intravenously and some vasopressor agent to increase the pressure, which has a tendency to fall while the operation is in progress. The most useful drug of this character is neo-synephrin hydrochloride, administered in doses of 0.5 cc. (5 mg.) either intramuscularly or intravenously.

Audiometric determinations were made in this series of 156 cases of hypertension, in 154 of which a diagnosis of essential hypertension was made while in 2 the diagnosis was malignant hypertension. Significant loss of hearing was demonstrated in 148 cases, or 96.1 per cent. The deafness, which was bilateral in nearly all cases, manifested a striking parallelism in the amount of loss of hearing and the tone frequencies involved in both ears. There seemed to be no parallelism between the duration of the hypertension and the degree of deafness.

An opportunity for further study of deafness in cases of hypertension was afforded in 37 cases of this series in which the patients submitted to splanchnicectomy for reduction of the blood pressure. Audiograms were made three months after the operation and compared with the preoperative charts. In most cases there was a striking decrease in the hypertension after the operation, and this was associated with some improvement in the hearing. However, it was not possible to demonstrate any correlation between the degree of improvement in the auditory function and the amount of reduction in the blood pressure after section of the splanchnic nerves.

Essential Hypertension from the Standpoint of Ophthalmology.
Dr. Henry P. Wagener, Rochester, Minn.

At this time the retinal pictures typical of chronic or rapidly progressive hypertensive disease have not been reproduced in characteristic form or course in experimental animals. In spite of the inadequacy of present experimental evidence, advances are being made in the understanding of the mechanism involved in the production of the retinal lesions and of their relation to the systemic, pathologic and pathologic-physiologic processes of the hypertensive person.

The conception that a toxic substance circulating in the blood is responsible for the lesions in the retina commonly designated as "retinitis" and that the kidneys are the sources of this toxic substance has been most widely accepted. It has been impossible thus far to demonstrate the existence of such a toxic substance.

Clinically, it seems obvious that the most severe forms of retinal vascular disease and of retinitis can develop in the course of hypertensive disease in the absence of any marked or even definite evidence of renal impairment. When severe renal insufficiency occurs in association with retinitis in patients who have primary hypertension, it seems probable that the damage to the kidneys is not the essential feature of the disease but that it is only a part of widespread injury to the vascular system.

In cases of "albuminuric retinitis" fairly widespread sclerosis of the arterioles has been demonstrated to be present in the retina at necropsy, but it seems unlikely, both from the clinical and from the histologic standpoint, that organic damage to the retinal vessels can be held responsible for the development of the forms of retinitis characteristic of hypertensive disease. It seems most logical at present to assume that retinitis develops in some manner as a form of decompensation of the retinal circulation and not from organic lesions in the walls of the vessels.

In general, the changes observed ophthalmoscopically to occur in the retinal arterioles in association with hypertensive disease may be grouped under five main subdivisions: (1) generalized smooth narrowing of the lumen, (2) changes in the color, reflex stripe and visibility of the walls, (3) arteriovenous compression, (4) irregular localized constriction of the lumen and (5) periarterial sheathing. In many cases various combinations of these changes are seen. Each of them can occur alone, however, and the significance of each should be considered separately. If the color changes consequent on changes in the blood itself are excluded, it seems probable that changes in the color of the wall of the vessel are due to an actual structural lesion in the wall, which is most often hypertrophy of the media. Irregularly placed, localized narrowings of the lumen of the arterioles have been demonstrated definitely in many instances to be of a transitory spastic, rather than of a permanent organic or structural, nature. The ultimate proof of the spastic nature of any localized narrowing rests, of course, on its complete disappearance. In rapidly advancing or active hypertensive disease, such as that seen in association with acute toxemias of pregnancy, shifting in the situation and degree of these localized regions of narrowing can be observed from day to day. As the hypertension becomes more fixed, the spastic narrowings may become more fixed in situation also.

In most cases, however, the size of the arterioles in the retina is most probably an expression of vasomotor tone, and reduction in their size below that of the average normal indicates an increase in their tonicity. That this is true is suggested by the fact that the diameter of the arterioles corresponds in a general way to the height of the diastolic blood pressure readings. Repeated observations in individual cases demonstrate that the size of the arterioles is not constant but may fluctuate with variations in the blood pressure. Thus, the caliber of the retinal arterioles may be seen to increase after splanchnic sympathectomy for the relief of hypertension.

The picture of localized groups of punctate exudates and hemorrhages, described under the name of "retinitis of arteriosclerosis," represents the end phase of venous obstruction or thrombosis, usually a feature of the cases of milder hypertension. It may also be seen in the terminal phases of "angiospastic retinitis," a term used to designate

types that have been known as "albuminuric." When acute angiospastic retinitis occurs in the presence of chronic sclerosis of the arterioles, the prognosis for the life of the patient is always more serious. Should a person have acute angiospastic retinitis with chronic sclerosis of the retinal arterioles, it means always that he has diffuse arteriosclerosis, no matter whether his primary disease is "essential hypertension" or "chronic nephritis." The retinitis can be regarded only as a manifestation of decompensation of the retinal circulation.

Essential Hypertension from the Standpoint of the Internist. Dr. Roy W. Scott, Cleveland.

In primary or essential hypertension there is a persistent elevation in both the systolic and the diastolic blood pressure, unrelated to primary renal disease, endocrine or vasomotor disorders. Goldblatt and his associates have clearly demonstrated the renal origin of arterial hypertension. Present day concept of clinical hypertension as presented in most textbooks on the subject may be summarized as follows: 1. Hypertension admittedly of renal origin is associated with glomerular nephritis, urinary obstruction, polycystic kidney disease and periarteritis nodosa with marked renal involvement and severe renal amyloidosis. 2. Essential hypertension is not generally believed to be of renal origin, because there is no demonstrable renal excretory insufficiency in the majority of cases; most patients with essential hypertension die of heart failure or of a cerebral accident without ever showing renal insufficiency; occasionally a case of essential hypertension is reported in which no anatomic evidence of renal arteriolar disease was exhibited.

Since Goldblatt's demonstration of the role the kidney plays in causing arterial hypertension, several cases of hypertension have been reported in human beings in which the renal artery was narrowed or completely occluded and in which on operative removal of the ischemic kidney the blood pressure returned to normal.

Diabetic Retinitis. Dr. Jonas S. Friedenwald, Baltimore (from the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital).

General Hypertension and Peripheral Optic Pathways. Prof. Dr. Joseph Igersheimer, Istanbul, Turkey (by invitation).

The pathologic processes in the trunk of the optic nerve and on the chiasma in cases of general hypertension (mostly in cases of malignant hypertension) are more frequent and more variable than one believes generally, and they warrant more attention.

Whether the alterations in the connective tissues, such as lymphocytic infiltrations around the central artery and in the septums or peculiar proliferations of the connective tissue in the axial fasciculus, are intimately connected with general hypertension must be left open to question. I have seen such alterations several times in different degrees, and they demonstrate that changes are taking place behind the globe which cannot be recognized clinically.

It is of more importance that degenerative processes in the optic nerve can also occur latently (without ophthalmoscopic signs). It is

probable that they cause sometimes, according to their situation and extension, disturbances of the visual functions. Furthermore, it is remarkable that retinal lesions of especially long duration by no means produce an ascendant degeneration but, on the contrary, just those forms which show clinically and anatomically few typical retinal spots. Instead of this, the small retinal vessels show high grade arteriolosclerosis with obliteration of their lumen and lead in this way to severe disturbances at the inner layer with involvement of the nerve fibers.

Moreover, it can be regarded as certain that processes in the optic nerve and in the chiasm may occur which are not dependent on the retinitis albuminurica. Retinitis and processes of the peripheral optic pathway can occur concurrently, as is evident especially in a long observed case of retinitis associated with bitemporal hemianopic scotoma.

It seems that such independent diseases of the peripheral optic pathways can occur in association with different clinical conditions, such as circulatory disturbances and glaucoma-like cupping, and, moreover, in the form of a rapid decrease of the vision combined with neuritic atrophy. Observations have been made which demonstrate these several possibilities, and in most instances clinical and anatomic studies have been made. It is deplorable that the manner of the development of these diseases is often still undiscovered. It is possible that the changes in the small vessels are of great importance, and some of the statements made can be considered from this point of view, but absolute proof of such a connection has not heretofore been obtained. It is is to be hoped that further systematic researches and favorable cases may lead to a more certain comprehension.

Acute Hydrops of the Cornea Complicating Keratoconus. Dr. Ralph O. Rychener, Memphis, Tenn., and Dr. Daniel B. Kirby, New York.

This article will be published in full, with discussion, in a later issue of the Archives.

Transplantation of the Cornea by Means of a Mechanically Obtained Segment with a Beveled Edge. Dr. Meyer Wiener and Dr. Bennett Y. Alvis, St. Louis.

Ever since transplantation of cornea has been seriously considered by ophthalmic surgeons, they have sought a mechanical means of obtaining a graft with a beveled edge. We have succeeded in designing a punch by means of which a circular segment of cornea having a beveled edge is punched out, the epithelial surface of which is about 0.6 mm. wider than the endothelial surface. The graft is not injured, as shown by macroscopic and microscopic observation. The punch is constructed so that the female blade is 6.5 mm. wide and 1 mm. thick. The upper surface of its bore measures 4.5 mm. in diameter. The under surface is wider, as the hole is cut on a bevel so as to make the upper cutting edge sharp. The male blade is bullet nosed, its diameter, 2.5 mm. from the tip, being the same as that of the hole in the female blade. When the sharp edge of the female blade, after being introduced into the anterior chamber, engages the under surface of the cornea, the tip of

the male blade presses down, reverses the curvature and stretches the entire anterior corneal surface, driving a wider portion of it into the punch. The graft is held in place by fine silk sutures properly placed and loosely tied. When the cornea of the host is vascular, an erythema dose of roentgen rays is given the following day and is repeated a second or even a third time if the vascularity seems to encroach on the graft.

DISCUSSION

DR. VIRGIL G. CASTEN, Boston: Corneal transplantation was first attempted about one hundred and sixteen years ago. In the early years, lack of both asepsis and anesthesia caused many failures. During the past century anesthesia and the technic of corneal transplantation have both greatly improved. Almost all the total corneal transplantations first done by Wagenmann in 1888, when the entire cornea and some conjunctiva were transplanted, resulted in failure.

Von Hippel in 1888 tried so-called circumscribed lamellar corneal transplantation, but connective tissue formed at the base of the transplant, preventing visual success. He then went back to his earlier method of 1877 of partially penetrating corneal transplantation, which Elschnig later found to be the most dependable method. Von Hippel used a trephine to cut his transplants.

Elschnig modified von Hippel's technic slightly and reported improvement in vision in many cases. Filatov further modified von Hippel's operation, making a puncture and counterpuncture of the cornea, through which a strip of celluloid was passed to prevent injury to the iris and lens when the cornea was trephined.

Thomas in 1930 used a 4 to 4.5 mm. trephine, held at an angle of 30 to 45 degrees, so as to penetrate at one side. The trephine was then withdrawn and the button completed with scissors. In this way the inner surface was made smaller than the outer, giving a beveled edge which prevented the transplant from falling into the anterior chamber.

Castroviejo in 1932 used a double-bladed knife, making a rectangular transplant, beveled on one side with a keratome and on the other three sides with scissors, as he found rectangular transplants easier to bevel than round ones.

All of these operations have been time consuming. Drs. Wiener and Alvis have ingeniously devised a new technic for securing uniform, mechanically obtained, beveled grafts, which can be cut in a second and transferred from donor to host. The average ophthalmic surgeon can cut uniform beveled grafts with this instrument. However, with this technic another 8 mm. corneal incision is necessary, which has not been used in any of the former operations. This should not be a great stumbling block.

In the way of criticism, it would seem to me the instrument could be used more easily if it had a bayonet end, so that the brow would not interfere, and also an infinitely thin sleeve to prevent the graft from falling into the anterior chamber when it is cut. Further, it would seem that the graft would be subjected to great trauma, and consequently there would be a higher percentage of opaque transplants by this method. This, however, remains to be seen, as only one operation has been reported.

The type of suture is excellent. Conjunctival flaps have more or less been given up because of the higher incidence of infections, and a suture of some type is being generally used.

DR. RAMON CASTROVIEJO, New York: Drs. Wiener and Alvis deserve to be congratulated for this ingenious instrument, which it is claimed will permit the average ophthalmologist to obtain mechanically a beveled graft.

In the operation described an incision is made at the limbus, through which one blade of the instrument is introduced into the anterior chamber. It is reasonable to suppose that this extra incision at the limbus will lead to complications, such as incarceration of the iris. This supposition is corroborated by the report of the only case in which this technic was used, and in this instance incarceration of the iris took place. Of course a single case is not sufficient from which to draw conclusions. Many more persons will have to be operated on, possibly several hundred, before definite conclusions can be drawn.

The condition of the anterior chamber must also be considered in performing keratoplasty with the technic of Drs. Wiener and Alvis. If the eye is aphakic, the used of the punch described may lead to luxation of the dissected leukoma into the vitreous cavity.

The simplified suture used by Drs. Wiener and Alvis is just one half of the cross stitches advocated by Tudor Thomas. I have observed, however, in the illustrations showing the different steps of the technic that the suture is placed too far from the edge of the incision. It has been the experience at the Institute of Ophthalmology, Columbia-Presbyterian Medical Center, where about 200 keratoplastic operations have been performed, that the sutures must be placed very near the edge of the incision; otherwise, in cases in which the cornea is extremely thin, especially in cases of keratoconus, the cornea collapses and it is difficult or impossible to maintain the transplant in position.

When the cornea is favorable for keratoplasty, a simplified stitch, such as the one advocated by Drs. Wiener and Alvis, may be sufficient. It is, however, good judgment to consider every case a difficult one, in order to avoid unpleasant surprises during the operation and complications during the postoperative course.

A beveled transplant seems to be obtained consistently and easily by the procedure described by Drs. Wiener and Alvis, and according to their statement the edges are cleanly cut, an observation confirmed by microscopic examination.

The ingenious operation presented here deserves to be thoroughly studied. A comparative study of this and of other methods of operation made on eyes of animals will give new data to report in the future as to the real value of the operation.

DR. B. Y. ALVIS, St. Louis: We realize that Dr. Castroviejo's last point is well taken, indeed. One case is not sufficient on which to draw any conclusion and much more study is necessary both to perfect the technic and to prove its value. This paper, however, was given rather as a preliminary report, because we felt that this contribution was sufficiently valuable in that it simplified the technic of corneal transplantation so much that it would be worth while to let others use it and attempt to perfect the technic along with us.

The simplicity of the technic, it seems to me, will make it possible for keratoplasty to be carried out far away from Dr. Castroviejo and others who have had more extensive experience with this type of operation. The length of time required for the operation is only fifteen or twenty minutes.

Some one inquired as to where the punch is made. It is made by the Storz Instrument Company.

The statement was made that the graft would be difficult to recover from the anterior chamber. Such has not proved to be the case. There were a few instances in experimental work that the graft did slip into the anterior chamber, but there was little difficulty in recovering it because of the large incision in the cornea. In fact, the graft is not often lost, and I do not believe that it is more likely to drop into the anterior chamber than if one used a trephine or the double corneal knife of Castroviejo.

The objection occurred to us that probably the graft would be crushed, but when one considers the cutting operation of this punch one notes that the margin is cut in exactly the same way as by the scissors. A microscopic study of such a section showed no visible evidence of crushing of the corneal tissue.

Relation of Vitamin Deficiency to Tryparsamide Reaction. DR. WILLIAM M. MUNCY, Providence, R. I.

The most satisfactory treatment of neurosyphilis is thought to be fever therapy followed by the administration of tryparsamide. As the fear of ophthalmic complications seems to be an objection to its use, a thorough study of each case before and during treatment seems most important. This investigation was carried out at the Charles V. Chapin Hospital of Providence, R. I. The findings in the first 40 cases were published in the *Rhode Island Medical Journal* (**20**:169, 1937). A reaction to this drug occurred in 6 of the cases, or 15 per cent. In 5 of these the patients had normal visual and form fields prior to the reaction, and there was nothing on ophthalmoscopic examination that would indicate any disease of the tissue viewed. On the other hand, among the other 34 patients were some with conditions such as old iridic adhesions, retinochoroiditis and even optic neuritis, who were given the drug without harm. From these findings I concluded that the previous state of the eyes was no indication of susceptibility to this drug. Furthermore, the fact that no objective signs were found on examination of the eyeground in these 6 cases during an attack led one to suspect that I was dealing with toxic amblyopia caused probably by vitamin deficiency. Two of the 6 patients who had adverse reactions were again treated with tryparsamide, after having been built up in health, and have had a number of series of treatments since without any untoward effects.

From the summer of 1937 to date, vitamin B and B complex have been given in a second series of cases, 50 in number, in which tryparsamide was administered. The vitamins were given constantly from the time of the patients' admission to the hospital, during fever therapy, throughout the series of treatments with tryparsamide and whenever the administration of the drug was resumed. A reaction occurred in only 1 of the cases. This was a case of atrophy of the optic nerve, probably due to retrobulbar neuritis; after tryparsamide shock, the

patient was brought back to his previous state by numerous intravenous injections of vitamin B. Since then the patient has undergone seventeen treatments with tryparsamide without any further reaction.

In addition, during this period a number of persons who had already had a reaction from tryparsamide were sent to the hospital for treatment. They had not received vitamins as a preventive treatment, but under active vitamin therapy their vision had been restored to its previous state. Among these there was a patient who had a secondary reaction when the administration of tryparsamide was resumed. This reaction followed the third treatment with tryparsamide in spite of numerous intravenous injections of vitamin B. There was a rapid loss of vision and form field in the left eye and complete blindness in the right eye at the time the patient died, twenty-one days after the last treatment. Autopsy disclosed marked arteriosclerosis throughout the brain, with numerous areas of anemia and soft degeneration. The extensive pathologic process found indicated that the cardiovascular disease had already reduced the life expectancy to a narrow margin at best.

These 2 patients were the only ones having had vitamins as a preventive treatment who reacted adversely to tryparsamide.

DISCUSSION

DR. W. I. LILLIE, Philadelphia: I presume that every one is in accord that necessary precautions should be taken concerning tryparsamide therapy. About 6 per cent of patients treated with this drug have untoward ocular reactions, and by far the larger portion present only the transitory symptoms, which disappear shortly after the use of the drug is discontinued. In a small percentage a progressive loss of vision develops, and evidence of simple atrophy of the optic nerve may appear as a late or final result. Various procedures have been instituted to prevent such occurrences, and the ophthalmologist has played an important role in the early recognition of impending visual damage. This impending visual change is usually first suspected by subjective visual complaints, and if the use of tryparsamide is discontinued promptly, the subjective symptoms usually disappear.

The oral and intravenous use of various vitamins have been of great value in many systemic diseases. The vitamin B complex has been of real value in some of the diseases that affect the central and peripheral nervous systems. To my knowledge, the preliminary use of the vitamin B complex to prevent any untoward visual reaction during tryparsamide therapy has been suggested and first used by Dr. Muncy. The comparative statistics on his two groups of patients are suggestive that the vitamin B complex played an important role in decreasing and practically eliminating the occurrence of any untoward visual reactions. The subsequent improvement of vision in his group of patients who previously had been affected during the tryparsamide therapy is not so significant, as vitamin therapy has been used frequently, and the results obtained are uniformly good comparable to those in Dr. Muncy's group.

My experience with vitamin therapy for ocular conditions has been so uniformly favorable that I do not hesitate to use the combination of vitamin B and C, orally or intravenously, as an adjunct to other accepted types of treatment. Inflammatory conditions of the retina, choroid and

optic nerve respond equally well, and improvement in the retinitis of diabetes and scurvy is sometimes startling. The treatment of either the primary or the secondary type of atrophy of the optic nerve with vitamins or any other therapeutic measure is usually of no avail, but the progressive visual deterioration can sometimes be slowed up or eliminated. At least the use of vitamin B and C, orally or intravenously, can do the patient no harm and may be of benefit. Its use is an addition to present day therapeutic measures.

Dr. Muncy is to be congratulated for having originated the idea of using vitamin B therapy as a possible preventive of the untoward ocular effects arising during tryparsamide therapy. I feel that ophthalmologists should establish a similar regimen, as suggested by Dr. Muncy, in their own institutions or practices, and later the sum total of individual experiences will answer Dr. Muncy's question: Does the preliminary use of vitamin B before tryparsamide therapy is instituted lessen or eliminate the occurrence of untoward ocular effects?

DR. WILLIAM M. MUNCY, Providence, R. I.: I agree with all that Dr. Lillie had to say, but I should like to emphasize the following points: The 2 patients in the first series who had an adverse reaction but were again given tryparsamide after they had been built up in health by a well balanced diet and had increased in weight were much slower regaining their lost visual fields than the later patients who were given vitamins intravenously. Furthermore, the failure in the last 2 cases was, I fear, due to overconfidence in the method being used.

The patient with atrophy of the optic nerve was given but 2 yeast tablets three times a day, and fever therapy was begun at once. Some writers hold that a high temperature dissipates vitamin B within the body. If so, the vitamin content of the body at this time was probably being decreased rather than augmented. Then to cap the climax, while the patient was still having high elevations of temperature, treatments with tryparsamide were started. It was notable that the active use of vitamins intravenously rapidly restored the patient to his previous condition, and when the administration of tryparsamide was again resumed, there was not a second reaction. If vitamin B complex is given by mouth, much larger doses should be used.

In regard to the patient who died, though his general condition had markedly improved and ocular examination showed as much advancement as one could expect, on going over the data since this paper was written it was discovered that other patients to whom tryparsamide was again administered had all gained in weight while this patient had lost weight during his stay in the hospital.

While Dr. Lillie was discussing this paper, a number asked me if I gave yeast cakes. Yeast tablets are not yeast cakes but concentrated vitamin B complex obtained from grains that are put out by numerous drug firms.

Treatment of Accommodative Convergent Squint. DR. WILLIAM THORNWALL DAVIS, Washington, D. C.

If amblyopia ex anopsia is present in cases of accommodative convergent squint, occlusion of the fixing eye is necessary until the vision

is 20/40. The vision of the fixing eye may then be reduced to this level by partial occlusion with an opaque lens. Alternation must be avoided.

The child must be taught to keep his eyes straight with vision blurred, leaving his hypermetropic correction off as much as possible. The vision will clear as he learns to dissociate his convergence and accommodation. He must be taught to distinguish between clear and blurred vision. Blurred vision means that the eyes are straight, and clear vision means that one eye is turned in. The child should practice this with the orthoptist until he understands and gets the "feel" of knowing every time he turns his eye in. He must form the habit of keeping his eyes straight without glasses. When he can read 20/20 with both eyes at the same time, eyes straight, there is no longer desire or need for him to squint for distant vision. When he can read Jaeger's test type 1 or 2 with the eyes straight and without glasses, there is no longer any reason for squinting for near vision.

The mother or nurse should be taught to give the child lessons at home on a visual acuity chart, without glasses and for near and distant vision. Bar reading is prescribed first with the glasses, and when this can be achieved it is tried without glasses.

Exercises in the clinic or the office are given on the synotophore, the diploscope and the Remy separator to teach the child to dissociate his convergence and accommodation, so that he can continue to do this after leaving the clinic. It takes an average of six months to one year to cure a patient of purely accommodative squint.

If there is a concomitant squint or other factor, such as hyperphoria or paresis of one of the ocular muscles, this factor obviously must receive appropriate treatment in addition to the treatment for the accommodative squint.

DISCUSSION

Dr. George P. Guibor, Ottawa, Ill.: The ophthalmologists here are indebted to Dr. Davis for emphasizing in this paper the type of strabismus which is most amenable to nonsurgical treatment. This type, which is practically universally called "accommodative esotropia," is the type in which the deviation is less for distant than for near vision. It is improved by the wearing of convex lenses, usually a + 3 diopter sphere with a cylinder, by the use of atropine and by fusion training.

Dr. Davis has emphasized, too, that squint is a symptom complex and that several etiologic factors are associated with the strabismus in most cases. In a similar way he calls attention to what he considers a cure for strabismus. There should not only be a cosmetic recovery from the deviation but also a functional recovery. Thus the visual axis should be parallel for distant vision and for near vision, and the fusion power should be normal. As a matter of fact, however, few normal persons have perfect fusion without fusion training. One should, therefore, not expect every patient with parallel visual axis to have perfect fusion ability. Most of them, however, have good binocular vision but many times have imperfect stereoscopic ability.

Like all those interested in motor anomalies, Dr. Davis reiterates that the most important factor in securing a satisfactory recovery from the deviation is the diagnosis of the type of squint. He also discusses the diagnostic routine and here, too, impresses on his readers the importance

of determining the degree of deviation for distant and for near vision with and without corrective lenses. He again repeats these measurements when the eyes are under a cycloplegia and in the different directions of gaze.

One interesting sentence in this paper especially attracts my attention. This sentence is as follows: "As glasses are a physical, economic and social handicap, one must know that other means are available for the correction of the squint or for the relief of symptoms." This refers to accommodative esotropia. This sentence to me suggests that orthoptic fusion exercises and surgical treatment can be used in place of glasses in treating this condition. I have attempted this several times but have always felt that I was not doing the correct thing by stimulating convergence indirectly through the accommodation; I feel that this is a more difficult method of treating neuromuscular anomalies than when accommodation is kept under control by glasses and atropine. But I shall have to revise my attitude concerning this routine of removing lenses and attempting fusion exercises when such men as Dr. Davis and those of the London school of orthoptics follow this technic.

In conclusion, Dr. Davis emphasizes the following causes for failure in treating accommodative esotropia: (1) severe heterophoria or anomalous position of rest coexisting with the squint; (2) vertical imbalance; (3) visual defects, such as amblyopia, and (4) anomalous retinal correspondence.

The following cases, which are briefly reported, emphasize my limited experience with accommodative esotropia associated with some of these causes for failure. Of course pure accommodative esotropia never presented these complications.

One patient had right esotropia of 50 degrees without glasses and 40 degrees with glasses for distant and near vision. I thought that the condition was due to a spasm of the right internal rectus muscle, and to overcome that spasm I employed a horizontal prism, base out, in front of the right eye. These prisms improved the esotropia so that it was only 20 degrees after several months. I should like to ask Dr. Davis what he does when he attempts to treat a patient with a spasm of convergence or a spasm of one internal rectus muscle or a spasm of accommodation by his method. One of the methods I have used in the treatment of accommodative esotropia associated with a spasm of convergence is to employ a prism, undercorrecting, of course, the degree of spasm as determined by the occlusion or screen test.

In another case an anomalous position of rest was demonstrated. The patient had 30 degrees of left esotropia without his glasses and 25 degrees with his glasses. After both external rectus muscles were dissected, he had an apparent parallelism of his visual axes. However, the point to be brought out with regard to the anomalous position of rest is that, though I did not touch the internal rectus muscle, under cover the patient at the present time has an exotropia of 10 prism diopters, which was brought about by the mere resection of both external rectus muscles. Thus this patient had a convergent squint with a convergence insufficiency, a condition that the ophthalmologist often fails to recognize.

As a third example, the effect of eccentric fixation in anomalous retinal correspondence on the result of nonsurgical treatment is demon-

strated by 2 brothers. One was 7 years of age and the other 13. Both boys had 25 degrees of right esotropia with glasses. The deviations of these boys do not vary more than 5 degrees or more than 10 prism diopters. The other interesting factor is that both of them wear similar lenses before their eyes. The older boy, however, who has normal correspondence, recovered after one year; the younger, who has anomalous correspondence and eccentric fixation, did not recover.

Thus the most common factors which produce failures in treatment are: (1) severe heterotropia coexisting with the squint, (2) a vertical deviation, (3) visual defects and (4) anomalous correspondence.

DR. GRADY E. CLAY, Atlanta, Ga.: Dr. Davis has stressed the most important factor in the treatment of accommodative convergent squint, namely, orthoptics. The treatment of this type of squint has progressed little during the past decade. For years the refractive error alone was corrected. Later some ophthalmologists attempted to obtain a cosmetic result by surgical treatment. Only within the past twenty years have most ophthalmologists insisted on surgical treatment as soon as it has been seen that glasses would not correct the squint. There are a few bold enough to insist on such treatment before any attempt is made to help the patient with a correction.

It is true that the parents of a squinting child are primarily interested in a cosmetic result, but as an ophthalmologist one should be most interested in the development of binocular vision, and I should like to ask how one can be sure of ever obtaining fusion without the use of orthoptic training. It is true, and most unfortunate, that the ophthalmologists who discredit orthoptics are those who have not earnestly sought its true value.

Most persons who develop fusion with good amplitude of accommodation have very blurred vision at first, which clears with training, but, in my opinion, to start the treatment of such a person with the whole idea of removing the glasses is not good practice. For persons with accommodative squint and small errors, this may be done easily. The child with accommodative squint and a small astigmatic error who is given a $+2$ diopter sphere, which straightens the eyes, can be trained to develop fusion with good amplitude in a short period, and thus may in the course of time leave glasses off without discomfort. Many of the cases which Dr. Davis has reported are those in which a high hyperopic astigmatic error exists. I do not think his procedure should be attempted in such cases. If it is adopted, it seems to me that there is no reason to give a patient a correction. However, for girls it is most important that this be attempted, and I feel sure that the large majority of them can be trained because of their interest in trying to develop fusion and to hold their eyes parallel without glasses. For this particular type of person I would certainly recommend that the effort be made, with the hope that the patient may, for social purposes only, leave her glasses off.

DR. WILLIAM THORNWALL DAVIS, Washington, D. C.: For a subject as intricate as this, it is utterly impossible to give the many, many

details. As regards surgical treatment of accommodative squint, it should not be used for a purely accommodative squint. It is absolutely contraindicated in such cases. If operation is performed the squint will be corrected but the patient will have an exotropia for distant vision and an esotropia for near vision. Perhaps I am mistaken, but I have had this unfortunate experience a great many times. Such persons should not be operated on unless another factor is involved.

Dr. Guibor asked about spasm of extraocular muscles and of accommodation. I have the feeling that a spasmatic action of one extraocular muscle must follow a paretic muscle and that there is another accommodative factor present. I may be wrong. My experience has been that it is dangerous, indeed, to operate in cases of anomalous retinal correspondence. In many cases the condition is incurable. If one disturbs the convergence system a wide swing in the opposite direction is likely to result and the patient will have a greater exotropia than esotropia, which cannot be corrected. I have learned that by bitter experience.

In regard to what Dr. Clay said about the use of glasses, I am entirely in sympathy with that. Of course, with high errors, particularly of astigmatism and spherical hypermetropia, the child will need glasses; but I was speaking of correction of the refractive error in regard to the squint only. I did not enter on the necessity of glasses for other purposes. Obviously, they may be necessary. What I meant to say was that one can cure accommodative squint associated with high refractive errors, so that it is not necessary for the patient to wear glasses to correct the squint. Perhaps I did not make that clear.

Of 98 persons with squint seen in the clinic of my associates and me from July 1938 to September 1939, 21 had accommodative squint. Seven are being treated now. The number whose treatment was incomplete due to insufficient cooperation is 6. The number considered corrected, that is, the number whose eyes are straight without glasses, is 8, or approximately 35 per cent. This is a rather low number, but one does not see many persons with purely accommodative squint. This is an important point. Of those included in the number whose squint was corrected, 3 had relapses two to three months afterward. It was necessary to institute active treatment again for these patients, and they responded promptly. Therefore, after treatment of accommodative squint a patient should be kept under observation for some time.

Visual Testing Methods in School. Dr. John B. Hitz, Milwaukee, Wis.

This article will be published in full, with discussion, in a later issue of the Archives.

Pulsating Exophthalmos. Dr. Samuel J. Meyer and Dr. H. Saul Sugar, Chicago.

This article will be published in full, with discussion, in a later issue of the Archives.

Retrograde Degeneration in the Optic Tracts and Nerves: An Experimental Study of Changes in the Axis-Cylinders.[3] Dr. P. J. Leinfelder, Iowa City.

Previous experiments indicate that after section of the optic tracts little or no degeneration occurs in the myelin sheaths of the optic nerves. However, after section of the optic nerve, complete retrograde degeneration of the myelin sheaths is observed. The present study of the axis-cylinders was made to determine whether the reaction in the axis-cylinders was similar to that in the myelin. Experiments were made on cats and monkeys; the results of previous experiments were confirmed, and it was determined that after section of one or both optic tracts retrograde degeneration was not present in the axis-cylinders of the optic nerves. When the optic nerve was sectioned at the chiasm, there occurred complete retrograde degeneration of the axis-cylinders and myelin sheaths.

The study also shows that the extent of retrograde degeneration is not a phenomenon determined by the distance from the lesion, for when the optic tract was injured at, or remote from, the chiasm, degeneration proceeded in a similar degree to the chiasm.

Further studies are directed toward determining a connection between the optic chiasm and the hypothalamus in an attempt to explain this difference in degeneration.

Glaucoma Following Roentgen and Radium Therapy. Dr. Louis Bothman, Chicago.

This article will be published in full, with discussion, in a later issue of the Archives.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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Oct. 26, 1939

Rhinosporidiosis of the Conjunctiva: Report of a Case. Dr. Harold J. Barnshaw and Dr. William T. Read Jr.

This article will appear in full, with discussion, in a later issue of the Archives.

Diplopia Following Fracture of the Malar Bone: Report of Cases. Dr. H. Maxwell Langdon and Dr. Robert H. Ivy.

Diplopia has been frequently observed following fracture of the malar or zygomatic bone involving the floor of the orbit. Thus in 11 cases of fracture this sign was noted in 4 by one of us (R. H. I). In only 2 was

3. This work is a portion of research studies in neuro-ophthalmology being made in the department of ophthalmology of the University of Iowa under a grant from the American Academy of Ophthalmology and Otolaryngology.

a detailed study made of the diplopia. In both it was found to be due to paresis of the inferior oblique muscle (H. M. L.).

CASE 1.—W. S., aged 17, seen on April 10, 1934, had been struck on the left cheek by a baseball two hours before. There was ecchymosis of the tissues of the left side of the face with some conjunctival hemorrhage. The globe was uninjured. The following day the swelling had subsided after the use of ice compresses, and there was found to be definite diplopia when the patient looked upward—a typical sign of weakness of the inferior oblique muscle. Fracture of the floor of the orbit involving the malar bone was diagnosed. The following day the depressed bone was elevated by one of us (R. H. I.), and the diplopia disappeared permanently and has not been present since. Central vision was never disturbed, and the patient's eyes are in other ways perfectly healthy.

CASE 2.—Miss C. C., aged 25, fell on Dec. 21, 1938, striking the right maxillary region on the stone edging of a driveway. There were much contusion of the tissues, swelling of the lids and subconjunctival hemorrhage. Central vision was not disturbed and the eyeball was healthy; however, when the patient looked upward there was double vision typical of a weakness of the right inferior oblique muscle. A roentgenogram showed a fracture through the outer portion of the lower rim of the orbit. The patient was seen on December 25 and operated on by one of us (R. H. I.) on December 30. The next day the diplopia was slight and in a week had disappeared, since which time it has not returned.

Comment.—The remarkable feature of the diplopia in these cases was that it cleared up almost immediately after the fracture had been reduced. The mechanism of the disability and of the rapid recovery is difficult to explain. The diplopia could hardly be due to actual trauma to the muscle or to infiltration with blood or serum, because in that case at least several days would be required for recovery. If it were due to injury of the third nerve, recovery would be delayed still longer. If one looks at the anatomic relations one notes that the inferior oblique muscle arises from the anterior inferior angle of the orbit and has no other bony attachments but passes outward and backward in the floor of the orbit to be inserted on the outer surface of the eyeball. Its bony attachment is a considerable distance medial to the usual fracture line, so that the paralysis cannot be explained by tearing of muscle fibers at the point of origin.

In the literature the nearest approach to this subject is found in a recent article by Novick, who has reported 2 cases of paralysis of the inferior oblique muscle following the Caldwell-Luc operation on the maxillary sinus. He stated that paralysis or paresis of this muscle is exceedingly rare and that few ophthalmologists or otolaryngologists have observed a case of isolated paralysis of the muscle. In his cases, Novick attributed the paralysis to injury of the muscle by penetration by the curet of the thin orbital wall of the antrum in the region of the origin of the muscle.

In our cases this explanation does not apply, because there could hardly have been any tearing of the muscle fibers by the injury, and also there would not have been such prompt recovery from the diplopia after reduction of the fracture. The only explanation we can offer for this temporary diplopia is a sagging or loss of tension of the inferior oblique muscle, which was instantly relieved by reduction of the fracture. In all

of our recent cases of fracture of the malar bone with displacement the method described by Gillies has been used. It consists in the use of a long elevator introduced through a small incision in the skin above the hair line, beneath the temporal fascia and the zygoma. By this means the depressed bone can be readily elevated into position even two or three weeks after the injury, and the operation leaves no visible scar.

DISCUSSION

DR. CHAPIN CARPENTER: I should like to inquire of Dr. Ivy as to the patient I saw for him at the Bryn Mawr Hospital two weeks ago. The picture presented by this patient was exactly similar to that in the cases just reported. When I saw the patient it was about forty-eight hours after the operation, and he still had diplopia. He left the hospital the next day, and as I have not seen him since I have been wondering whether the diplopia cleared up.

DR. ROBERT H. IVY: I saw the patient mentioned by Dr. Carpenter this morning. I did not examine his eyes or test him for diplopia, but he said that the double vision had disappeared. The most important point about these cases is the necessity of early replacement of the depressed fragment in order to avoid permanent disfigurement. After three or four weeks the bone may become firmly fixed, and elevation is often impossible; replacement is usually simple if done in the first two weeks.

Management of Some Complications of Cataract Operations. DR. EDMUND B. SPAETH.

A series of complications following cataract extractions, including severe traumatisms, postoperative infections, subchoroidal hemorrhages, sympathetic ophthalmia, iritis with secondary glaucoma and prolapse of the iris are presented to illustrate certain important points in their treatment.

DISCUSSION

DR. FRANCIS HEED ADLER: This is an instructive paper, as one can usually learn more by hearing of the complications of cataract extractions than of the excellence of the results. The most annoying complication which I encounter is that of incarceration or prolapse of the iris. It does not seem to make much difference whether one does a complete or a peripheral iridectomy, prolapse sometimes occurs; when it is large it is difficult to handle.

I should like to ask Dr. Spaeth what his experience is with prolapse and if he knows of any method by which is can be prevented.

DR. WALTER I. LILLIE: Any one who performs operations on the eyes certainly encounters the complications that have been discussed by Dr. Spaeth. The use of sutures at operations for cataract minimizes the chances of severe prolapse of the iris. Also the use of a pressure bandage rather than a pad and a shield has, in my experience, lessened the occurrence of prolapse.

DR. ALFRED COWAN: I have examined a great many eyes after cataracts have been extracted, the operations having been performed by a number of surgeons. I think that there is hardly a case in which either prolapse of the iris or anterior synechia does not occur, regardless of the procedure used by the surgeon.

DR. LEIGHTON F. APPLEMAN: I have been wondering whether after the patient's eyes have been bandaged and he has been returned to bed he may not have a feeling of irritation which provokes squeezing of the orbicularis ciliaris in such a way as to produce pressure on the globe, causing the lips of the wound to open slightly and thus allowing the iris to slip forward and become caught between the lips of the wound. With this in mind, I have been particularly careful to warn the patients beforehand to avoid doing this during the time of healing.

DR. CARROLL R. MULLEN: With knowledge of the experimental work which had been done to prevent prolapse of iris, I performed a series of simple intracapsular cataract extractions with the use of corneoscleral sutures. Suturing consisted of a transverse passage through the cornea and a return transverse passage through the scleral or episcleral tissue, so that a double "lock" would close the operative incision. This was performed at the Wills Hospital. Instillations of solutions of paredrine (4-hydroxy-α-phenyl-β-aminopropane; β-4-hydroxylphenylisopropylamine) and homatropine hydrobromide were given an hour before the operation in order to obtain wide dilatation of the pupil. Intraocular tension was recorded previous to the use of this medication, immediately preceding the retrobulbar use of procaine hydrochloride and about two minutes after the latter injection. Care was taken that there was no prolapse before the patient left the operating room. A weak solution of physostigmine salicylate was instilled immediately after the operation in order to bring about more favorable contraction of the pupil. In my series of approximately 30 to 35 cataracts, I was dismayed with a 30 to 35 per cent occurrence of prolapse of the iris. The late operations were just as prone to this complication as were the early experiences. Needless to say, I have abandoned the simple extraction.

DR. LOUIS LEHRFELD: Many complications following cataract extractions may be prevented by careful study of the patient prior to operation. If the surgeon is aware of the fact that he has an uncooperative patient, he must be prepared to select anesthesia which will enable him to keep that patient under complete control during the operation. It makes no difference whether the anesthesia is general (ether or rectal anesthesia with avertin with amylene hydrate) or local (combination of procaine hydrochloride and cocaine hydrochloride). I feel that the complete anesthesia at the time of the operation will help considerably in preventing such complications as prolapse of the iris.

At the Wills Hospital I give routinely a retrobulbar injection of a 2 per cent solution of procaine hydrochloride, a subconjunctival injection of a 1 per cent solution of cocaine hydrochloride in the superior rectus muscle and another subconjunctival injection of cocaine hydrochloride at the point where the fixation forceps is applied to the eyeball. Local instillation of a 4 per cent solution of cocaine hydrochloride, as generally used by all surgeons, is likewise a part of the procedure to obtain complete anesthesia.

The patient is thus under complete control; in this way unnecessary and unexpected movements of the eyeball are avoided, making the cataract extraction possible without complications on the operating table. It also permits complete rest of the patient for several hours after the cataract extraction.

Corneoscleral sutures, as introduced by me at the Wills Hospital, have also served to prevent complications by permitting patients to get out of bed shortly or soon after the operation. I have had a number of patients who were permitted to sit in a rocking chair immediately on returning from the operating room and were allowed to go to bed only to sleep at night. In one particular instance, a stout woman, weighing approximately 300 pounds (135 Kg.), was so treated without complications. Other instances can be cited among the aged, who were treated in the same manner. Other patients are routinely permitted to get out of bed on the third, fourth or fifth day, depending on the individual circumstances. By this method the surgeon avoids the postoperative tympanites, backache, restlessness and tampering with the dressings.

Complete anesthesia of the eye and the use of corneoscleral sutures (or what I prefer to call corneoepiscleral sutures) are both instrumental in the prevention of postoperative complications.

Dr. William Zentmayer: Some reference has been made to the effect of bandaging on prolapse of the iris. I have not used a bandage after cataract operation for the last fifteen or twenty years, but have used instead narrow strips of isinglass plaster over the closed lid, running from brow to cheek, the only protection being a mask. The roller bandage which is applied around the head is objectionable. The disturbance of the bandage produced by movements of the head is transmitted to the pad in front and disturbs its position.

The only objection to the strips is that they cannot be made sterile, but I have never had infection from them. I do not know that my proportion of prolapses is any less than that of the average operator, but I feel that a bandage might be the cause in some of the cases.

Dr. Edmund B. Spaeth: There will be as many different decisions, opinions and recommendations in regard to this problem as there are ophthalmologists present. I cannot agree with some of those stated. In many instances sutures do prevent a prolapse of the iris, but prolapse does not occur until twenty-four to forty-eight hours after operation. The effects of the anesthesia are gone, the aqueous is reforming and anything that will interfere with the free passage of aqueous through the pupillary aperture from the posterior chamber into the anterior chamber will cause prolapse of the iris. It is for this reason that peripheral iridectomy is actually more efficacious in preventing prolapse of the iris than is complete iridectomy, for the peripheral iridectomy lies at the root of the iris. As Dr. Cowan said, the all too common finding of anterior synechia in the iridocorneal angle following complete recovery shows plainly the underlying cause in its formation. I know of no measure which can prevent it except that mentioned (the peripheral iridectomy and corneoscleral sutures) when preceded by an adequate toilet of the wound and by the all-important first forty-eight hours of uneventful convalescence.

Clinical and Experimental Studies of Interstitial Keratitis. Dr. Joseph V. Klauder, Dr. Elmer R. Gross and Dr. Harold F. Robertson.

The following theories have been advanced for the motivating factor in interstitial keratitis: (*a*) that it is caused by direct invasion by

Spirochaeta pallida, (*b*) that it is due to an allergic reaction and (*c*) that it is due to a nutritional disturbance.

Sections of cornea removed from patients with interstitial keratitis failed to show S. pallida when examined by different methods.

Rabbits were given intraocular injections of horse serum. The ocular response to the second injection was studied by the slit lamp. There was no definite evidence of an allergic reaction. Pericorneal congestion and contraction of the pupil, however, occurred.

Cutaneous tests were performed on patients with interstitial keratitis, a piece of cornea of the same patient and also the cornea of a syphilitic fetus being employed. The reactions were negative.

It was not possible to produce interstitial keratitis in syphilitic rabbits by repeatedly traumatizing the cornea. The slit lamp picture of interstitial keratitis was produced in some normal and in some syphilitic rabbits by intracorneal injections of horse serum. This was attributed to trauma.

Studies were made of the effect of heat applied to the cornea in the treatment of interstitial keratitis. These embraced (*a*) the use of the thermophore (an instrument used to heat the cornea); (*b*) the measurement of the degree of heat penetration of the cornea of rabbits by the thermocouple; (*c*) the determination of the thermal death point of S. pallida in relation to the maximum temperature at which the cornea can safely be tested, and (*d*) the clinical effect of the thermophore in the treatment of the involved as well as the uninvolved eye of patients with interstitial keratitis and in the treatment of interstitial keratitis in syphilitic rabbits.

From these studies it was shown that the cornea is a poor conductor of heat. When the thermophore at 130 F. (54.4 C.) was applied to the anterior surface of the cornea, the maximum temperature of the posterior surface behind the thermophore was 111 F. (43.9 C.). The maximum temperature which the cornea will tolerate with safety is 130 F. (54.4 C.) for one minute. The temperature required to kill S. pallida was at least 120 F. (48.9 C.) for two minutes. It is therefore not possible after employing the thermophore in the treatment of interstitial keratitis to conclude by deduction that S. pallida are not present in the cornea.

Absorption of neoarsphenamine by the cornea after instillation into the conjunctival sac and the penetration of the drug into the cornea after intravenous administration in rabbits were studied. No arsenic was present in the cornea after successive instillation of neoarsphenamine in a dilution of 1:600. After six consecutive daily intravenous injections of neoarsphenamine, 30 mg. per kilogram of body weight, arsenic was present in each cornea in the amount of 0.04 mg.

The involved and uninvolved eye of patients with interstitial keratitis were treated with repeated instillations of 9 cc. of neoarsphenamine in a dilution of 1:600. This solution was maintained in the sac for twenty minutes. Such treatment did not influence the course of interstitial keratitis.

This article was published in full in the *Journal of Investigative Dermatology* (**2**:157-173 [Aug.] 1939).

Book Reviews

Primer of Allergy. By Warren T. Vaughan, M.D. Price, $1.50. Pp. 140. with numerous illustrations. St. Louis: C. V. Mosby Company, 1939.

It is timely that Dr. Vaughan, who is well known for his large book entitled "Practice of Allergy," which is intended for the medical practitioner, should also undertake to write a primer in simple language for the lay patient.

The work is divided into a number of chapters, with a summary and questions and answers at the end of each chapter, which makes the subject matter comprehensible. The illustrations are amusing, as a number of the well known cartoons of Webster have been introduced. An important part is the last chapter on general orders and directions to the patient, which contains much necessary information.

ARNOLD KNAPP.

The Journal of Endocrinology. Price, $6 a year. Published quarterly. London and New York, Oxford University Press.

This new journal will bring together the papers written by British investigators on endocrinology in one publication, which will be solely devoted to endocrinology. It is to be conducted by a council of management. Prof. E. C. Dodds is the editor. He is assisted by an editorial board composed of the following members: Dr. P. M. F. Bishop, Prof. C. D. Harrington, Prof. G. F. Marrian, Dr. A. S. Parkes, Dr. F. G. Young and Dr. S. Zuckerman. Dr. R. L. Noble has been appointed as assistant editor.

The first number appeared June 1, 1939, and contained eleven original articles. The journal will be published quarterly, and four issues will constitute one volume.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6°, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6°, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 4-6, 1940.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: New York. Time: June 10-14, 1940.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cleveland. Time: Oct. 6-11, 1940.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

Association for Research in Ophthalmology, Inc.

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

Canadian Medical Association, Section on Ophthalmology

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.
Place: Royal York Hotel, Toronto. Time: June 19-21, 1940.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
Executive Director: Mrs. Eleanor Brown Merrill, 50 W. 50th St., New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. G. Nadeau, 122 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: The Gateway Inn, Land O'Lakes. Time: June 1940.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick G. Sprowl, 421 Riverside Ave., Spokane, Wash.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Spokane, Wash. Time: June 24-27, 1940.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. A. Shultz, 303 N. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
Secretary: Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Virgil Payne, Pine Bluff.
Secretary-Treasurer: Dr. Raymond C. Cook, 1005 Donaghey Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Melville Black, 424 Metropolitan Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Shirley H. Baron, 309 State St., New London.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St. N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. D. C. Montgomery, 301 Washington Ave., Greenville, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: St. Charles Hotel, New Orleans. Time: April 25, 1940.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. O. B. McGillicuddy, 124 W. Allegan St., Lansing.
Secretary: Dr. A. R. McKinney, 330 S. Washington Ave., Saginaw.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Hendrie W. Grant, 330 Lowry Medical Arts Bldg., St. Paul.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. James S. Shipman, 542 Cooper St., Camden.
Secretary: Dr. Wright McMillan, 23 Passaic Ave., Passaic.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. J. W. Ward, 2607½ Lee St., Greenville.
Secretary: Dr. Dan Brannin, Medical Arts Bldg., Dallas.
Place: Fort Worth. Time: December 1940.

Utah Ophthalmological Society

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Harry M. Sage, 9 Buttles Ave., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Tex.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. Charles Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE AND EAR SOCIETY

President: Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, Brown Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Herbert G. Smith, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. David H. Webster, 140 E. 54th St., New York.
Secretary: Dr. Robert K. Lambert, 10 101–15th Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven, Conn.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President. Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Matthew Hosmer, 384 Post St., San Francisco.
Secretary: Dr. Fred Boyle, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. I. Henry Smith, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter W. Henderson, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.
Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.